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SPONTANEOUS PNEUMOTHORAX

IN

THE APPARENTLY HEALTHY

BY

HANS KJÆRGAARD

København

LEVIN & MUNKSGAARD PUBLISHERS
COPENHAGEN · MCMXXXII

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Denne Afhandling er af det lægevidenskabelige Fakultet antaget til offentlig at forsvares for den medicinske Doktorgrad.

København, den 9. Oktober 1931.

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PREFACE

The studies here reported*) were commenced at the time I was Assistant Physician to the Medical Policlinic of the Rigshospital, University of Copenhagen. They cover a group of patients from Copenhagen and from the country.

This work would have been impracticable to me if I had not met with — *everywhere* — a most generous goodwill and helpfulness.

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H. K.

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INTRODUCTION

Pneumothorax is a secondary lesion, that may be due to many different conditions, but as a rule the underlying disease will be easy to make out.

Now and then, however, one will meet with cases in which the cause cannot be ascertained with our present diagnostic means. These are the rare cases in which a person, who is feeling perfectly well and has a past history of good health, gets suddenly a spontaneous pneumothorax — cases, where even the most painstaking and thorough examination fails to reveal the cause of the lesion, so that the pneumothorax of the patient appears as the disease itself.

It is this spontaneous pneumothorax in apparently healthy individuals that is treated of in this study. The principal features in the clinical picture of this lesion are:

1) that it is a spontaneous pneumothorax, making its appearance in perfectly healthy young persons in whom neither emphysema nor tuberculosis may be demonstrated;
2) that it takes a benign and afebrile course, without any formation of exudate;
3) that the disease has a pronounced tendency to spontaneous recovery.

The records of these cases are so strikingly concordant that one needs to have observed the progress of this lesion but a couple of times in order to realise what Trousseau calls "la marche naturelle des maladies".

It is this unquestionable parallelism that points towards

a common cause. But, what this cause may be, that is a question it has been hitherto very difficult to have any clear idea about, because the patients practically always survive the lesion so that there is no opportunity to make an anatomical examination of the lungs in these cases. Up till now there has been reported a total of only 6 cases (Ranking, Pitt, Hayashi (2 cases), Brunner, Fischer), that have been fully cleared up on autopsy; and as only two of these reports date back before 1915, the cause of this lesion was until that time almost entirely a matter of hypothesis.

Quite naturally, then, the various authors differ considerably in their view as to the etiology of spontaneous pneumothorax in apparently healthy individuals:

1. Some authors think that these cases are due to a *latent non-demonstrable tuberculosis* (Mc. Kie (1905), Cummer (1915), et al.). Reiche (1918) and Dorendorf (1926) take this as the most probable causation; and Pierre Labro (1922) thinks that this form of spontaneous pneumothorax is often the first sign of pulmonary tuberculosis.
2. Some authors think that the lesion is due to a *rupture of an emphysema vesicle on the basis of a small tuberculous affection* (Brünniche (1856), Ljungdahl (1918)).
3. Others think that it is due to the *rupture of a simple emphysema vesicle* (Mc. Dowell (1856), Galliard (1888), Klemperer (1893), Goodhard (1896), Levison (1899), et al.). Besides, there are some authors who designate the lesion merely as *non-tuberculous pneumothorax* (Fussell & Riesmann (1902), Cahn (1917), le Wald (1927), Friesdorf (1927)).
4. Some authors think that the clinical picture of the lesion covers cases of *diverse etilogy,* latent emphysema as well as latent tuberculosis. This view is advocated by West (1897), Lindhagen (1915), and Hamman (1916).

5. In later years, J. Hayashi (1915) and B. Fischer (1922) have demonstrated that in 3 cases which they examined anatomically, the pneumothorax was due to the *rupture of a solitary air vesicle, developing from scar tissue and closed by a valve-like mechanism, at the apex of the lung.*

Since Mc. Dowell, in 1856, as the first described a clear-cut case of spontaneous pneumothorax in apparently healthy individuals, about 200 cases have been reported in the literature.

In 1856, the Danish physician A. Brünniche described a case of spontaneous pneumothorax in an apparently healthy young man with quick recovery. But, as this was associated with a small pleural exudate, it cannot be taken as a pure case of this lesion. Brünniche advanced the view, that this case might probably be due to a rupture in an area of emphysema produced by tuberculosis.

More extensive studies on spontaneous pneumothorax in apparently healthy persons have been published by Hall (1887) and Galliard (1888) and, later, by Nikolsky (1912) and Enneking (1923).

In the Scandinavian countries several interesting papers have been published on this subject: in Denmark by J. Liisberg and, more recently, by Emil Als, Erik E. Faber, Rudolf Kiær, Otto Lassen, and G. E. Permin; in Norway by Ed. Bull; in Sweden by Lindhagen, Ljungdahl and Dahlstedt & Haeger; and in Finland by Tallquist, Johannsen, and Fogelberg & Wallgren.

The most significant advance in the understanding of spontaneous pneumothorax in apparently healthy individuals has been made through the aforementioned studies of J. Hayashi and his chief, B. Fischer. From their autopsy findings in 3 cases in which a very thorough macroscopic and microscopic examination was made, these authors claim that the lesion is owing to the rupture of solitary air vesicles, closed by a valve-like mechanism and located in

scar tissue at the apex of the lung in persons whose lungs are otherwise perfectly healthy.

Up till now, however, this view of the etiology of the lesion has been based only on the few cases in which an anatomical examination has been made, and there has not been any clinical basis sufficient to apply this interpretation to all cases of the disease. Considering the above-mentioned variance of opinions advocated by the various authors, it is therefore not surprising that text-books of intern medicine are very guarded in dealing with this lesion (cf. Laache (in "Nordisk Lærebog i Intern Medicin") and Strümpell's "Spezielle Pathologie und Therapie").

This obscurity was the reason why I tackled this question when the occasion presented itself in form of two patients with this rather infrequent disease who simultaneously applied for treatment in the Medical Policlinic of the Rigshospital, Copenhagen. In meeting these two cases of pneumothorax I felt very keenly the difficulty of deciding on the treatment and making the prognosis, for the medical knowledge so far could not answer decisively the question, whether the clinical features of this lesion covered cases of one etiology or another (emphysema and tuberculosis), or whether they might constitute in themselves a nosographic unity.

It has been pointed out already that our anatomical knowledge of spontaneous pneumothorax in apparently healthy individuals is based on a total material of only 6 cases. But, it is also to be emphasized that the clinical aspects of the lesion are so far elucidated but insufficiently, and for several reasons.

For one thing, a large part of the reports on this lesion have been published before the introduction of X-ray examination, or they fail to give any information as to this point. In the next place, the lesion is so rare that *even though ca. 200 cases have been reported, nearly all the literature on this subject consists in scattered single cases.* Up till now, no fairly large number of cases have been

presented by one author, the greatest number reported by one author being 8 cases. It is true that Korach states he has seen 10 cases of this lesion, but he does not describe them in detail. Finally, most of the cases have been published shortly after the illness, and only a very few authors have given any data on the condition of the patient later on. Add to this the indistinctness in the conception of the clinical picture of the lesion that naturally results from the lack of harmony amongst the authors with regard to the question, which cases ought to be included in the clinical picture of this lesion. It is very important that this last point is made clear, and it will, therefore, be discussed in a subsequent chapter on the definition and delimitation of the clinical entity of the lesion.

I shall now give an account of my material and my working method. *The material consists of 51 patients with 61 attacks of pneumothorax* which, through the obliging courtesy of fellow-physicians, I have been able to gather, partly from Copenhagen and partly from various localities here in Denmark. These cases have occurred between May 1909 and July 1929. 34 of these cases have been under hospital treatment in Copenhagen, 11 have been in hospitals outside Copenhagen, and 6 cases have kindly been left to me by practising physicians in the country and in Copenhagen. The 11 cases from country hospitals have been collected on application to ca. 50 hospitals all over the country, while amongst the practising physicians I have applied only to those I knew had been treating such cases.

I wish at once to emphasize that I have made no selection of the cases whatever. Every case I have had access to, which falls under this heading, has been included in the material here presented.

In 1928—29, I have further looked up these patients to make an after-examination of them. At this, a history is taken of each case, covering the entire intermediate

period, a thorough auscultatory examination is made, and the lungs are roentgenographed.

In 1930—31, the after-examination is repeated on several of the patients in order to supplement the previous findings.

Finally, in 1930, I have made some pathologic-anatomical studies on suitable material to throw some light on the etiological basis of this lesion.

In the work here presented I have aimed, by means of this material of 51 patients, to give a contribution to the clinical understanding of spontaneous pneumothorax in apparently healthy individuals.

ETIOLOGY AND PATHOGENESIS

Undoubtedly it is impossible that a normal pleura might rupture through coughing or muscular exertion. West[2] has made some experiments with inflation post mortem of the lungs in situ, and he found that a rupture of the pleura could not be produced till the pressure was above 200 mm. Hg; and such a great intrapulmonal pressure can never arise in life. So one must always assume the presence of some pathological condition at the site of the pleural rupture as prerequisite.

Patients with true chronic emphysema of the lungs may have spontaneous pneumothorax, but it happens very seldom, although emphysema is quite a common disease. In Biach's statistics of 918 cases of pneumothorax, emphysema of the lungs is put down as the cause in only 7 cases. This finding is rather surprising in view of the marked atrophy of the lung tissue in emphysema. In fact, distension and atrophy of the lung tissue is the anatomical characteristic of emphysema, no matter whether this lesion is the result of chronic bronchitis, (Laënnec's "catarrhe sec"), or it develops through a deformity of the thorax in kyphosis (Loeschke). To what extent the lungs may undergo atrophy im emphysema, is evident from the measurings of Zahn. He found that while the thickness of alveolar wall + pleura is normally 0.13 — 0.24 mm., in emphysematous lungs it may diminish to 0.05 — nay, even to 0.03 mm.

The first case of spontaneous pneumothorax resulting from emphysema of the lungs was described by Devilliers, in 1826. Such cases have been reported subsequently by

Church. E. Winge, Mackenzie, Zahn, Stevens, Orth, and Bach. Bilateral spontaneous pneumothorax from an extreme degree of emphysema has been described by Massini & Schönberg, Alfred Meyer, and Emerson & Beeler. But in all these cases it has been evident from the history (chronic bronchitis, asthma) as well as from the physical examination, that the patients have suffered from pronounced emphysema of the lungs, which gives an anatomically and clinically different form of spontaneous pneumothorax that must be distinguished completely from the form of spontaneous pneumothorax which occurs in apparently healthy persons (cf. p. 42.).

Pulmonary tuberculosis is the most frequent cause of spontaneous pneumothorax, which sets in most often in the late stages of this disease when large parts of the normal lung structures have been destroyed, although in rare instances it may make its appearance in the initial stage of the phthisis.

So, a priori, one might very well imagine that a rupture of a minimal subpleural tuberculous focus might be the cause of the spontaneous pneumothorax in the apparently healthy individuals under discussion. For one thing, this view might be suggested by the fact, that in consumptives one will sometimes meet with an instance of spontaneous pneumothorax which takes a benign course, without any exudate. Further, in rare cases a minimal tuberculous focus has been demonstrated as giving rise to a spontaneous pneumothorax.

As a rule, however, when a patient with pulmonary tuberculosis has spontaneous pneumothorax, it takes the form of sero- or pyopneumothorax, owing to infection of the pleural cavity. This happens in all the cases due to rupture of a caseous focus. Yet, in rare instances, a consumptive may have spontaneous pneumothorax through rupture of an emphysematous vesicle in an area of vicarious emphysema; and this is not associated with pleural effusion. Such a case, with post-mortem examination, has

been reported by A. Brünniche as early as 1856, while Rheder has published a similar case in 1866; and the findings of these authors have later been confirmed by others. Recently, Möllerström has reported such a case, with microscopic examination of the site of the rupture. Unfortunately, in the literature one cannot find any data on the frequency of spontaneous pneumothorax of this origin in patients with pulmonary tuberculosis.

Cases in which a consumptive has had a dry spontaneous pneumothorax that recovered, have been reported by Guttmann, Wagner, West[4], Lucius Spengler[1,3] and others. Probably all such cases of benign spontaneous pneumothorax in consumptives are owing to a rupture of an emphysematous air-vesicle.

As mentioned, another reason why one might imagine the spontaneous pneumothorax in apparently healthy individuals as being due to a condition of latent tuberculosis, is the fact that there are published a few, rare, instances where a minimal tuberculous focus has given rise to spontaneous pneumothorax. But, in these cases the clinical picture has been entirely different from that which is typical of spontaneous pneumothorax in apparently healthy persons. A pleural exudate was present in Thomson's case (1839), and also in the case reported by Flint (1867). In the case reported by West[2] (1887) the patient died from exhaustion; and the case of Letulle (1901) was complicated which bronchiectasis, fever and pleural effusion.

So, in all the literature, as far as I have been able to find out, there is not one single instance in which the rupture of a tiny tuberculous focus has been the cause of spontaneous pneumothorax in an apparently healthy person, taking the course that is typical of this lesion and unaccompanied by fever and pleural effusion.

The chief source of information as to the etiology of spontaneous pneumothorax in apparently healthy individu-

als is found in the 6 cases of this lesion, in which autopsy was performed on account of special conditions.

A survey of these cases shows:

1. *Ranking* (1860):

Clinical data: Ordinary, mild, case of pneumothorax on the left side in a man, aged 19, with a past history of good health. Some time later the patient died from a dissecting aneurysm of the aorta, and the lungs were examined post mortem.

Autopsy: The left lung shows a few distended emphysematous vesicles, especially near the apex, but no other abnormalities. No sign of tuberculosis.

2. *Pitt* (1900):

Clinical data: Hæmopneumothorax on the right side in a man, aged 18, with a past history of good health.

Autopsy: Right-sided hæmopneumothorax (ca. 4,5 liters non-coagulated blood in the right pleural cavity). Near the apex of the right lung is a small, protruding, emphysematous bulla, fully 1 cm. in diameter. There is a defect in the wall of this vesicle. Attached to the vesicle is a ruptured pleural adhesion, about as thick as a knitting needle. Presumably the bleeding has come from this adhesion.

3. *Hayashi* (1915):

Clinical data: Woman, aged 60, with latent chronic pneumothorax, which is revealed on autopsy. The patient died after an operation for sarcoma of the stomach.

Autopsy: Pneumothorax on the left side. On the surface of the left lung, near the apex, and next to an adhesion, is a distended spherical emphysematous vesicle, wherefrom a small round hole leads into the lung. The upper part of the superior lobe is occupied by dense, anthracotic, connective tissue with a small calcified focus; the lower part of this lobe is greyish-black in colour and atelectatic. There is also a small anthracotic scar in the inferior lobe.

Also the right lung shows anthracotic scars and small calcified foci.

4. *Hayashi* (1915):

Clinical data: Fatal case of bilateral spontaneous pneumothorax in a man, aged 19, with past history of good health.

Autopsy: Bilateral spontaneous pneumothorax. The apices of the lungs show several scars; between these scars the surface is studded with groups of elevated, thin-walled, emphysematous air-vesicles, ranging in size from lentils to cherries. At the left apex, a tiny round opening is found in one of these emphysematous vesicles, which has the size of a hazel-nut. The right apex is adherent to the chest wall with cord-like adhesions, and next to these is a group of emphysematous vesicles. No sign of tuberculosis, neither macroscopic nor microscopic.

5. *Brunner* (1921):

Clinical data: Woman, aged 30, with chronic pneumothorax on the left side, which has lasted 4 years, and has lowered her working capacity very much. She has an attack of acute influenza-pneumonia on the right side, terminating in death.

Autopsy: Pneumothorax on the left side. No signs of tuberculosis or emphysema. The left apex is adherent to the chest wall with several adhesions (about the thickness of knitting needles). Next to these adhesions are a number of bullous emphysematous vesicles; and one of these shows a small perforation, hardly 1 mm. in diameter. The right lung shows an area of pneumonia.

6. *Fischer* (1922):

Clinical data: Hæmopneumothorax on the right side, terminating fatally, in a man, aged 22, with a past history of good health, as far as the lungs are concerned. As the patient has been suffering from attacks of dyspepsia, and

as the examination shows marked tenderness and board-hard muscle tension over the upper right part of the abdomen, his condition is first taken to be perforation of a duodenal ulcer, and explorative laparotomy is performed.

Autopsy: Right-sided hæmopneumothorax (ca. 4,5 l. blood). The lungs show no abnormalities, with the exception of some small, scar-like, retracted areas over both apices with multiple, greatly protruding, vesicles. One of the larger vesicles at the right apex shows a small rupture; the edges of this rupture are covered with fresh coagula. The basis of the vesicles is formed by the scar-like connective tissue. Otherwise, the lungs appear to be perfectly normal, showing no signs whatever of tuberculosis or emphysema, neither macroscopic nor microscopic.

Although there are not any more than these six cases that have been fully cleared up, they show such a uniformity of salient features that this can hardly be accidental. *In each of these cases the cause of the pneumothorax is found to be a rupture of a solitary vesicle at the apex of the lung, with or without scar tissue.*

Still, spontaneous pneumothorax in apparently healthy individuals is not always due to rupture of a demonstrable air vesicle. Both Rolleston and Rudolf Kiær have each described a case of fatal hæmopneumothorax where the autopsy failed to reveal any cause of the lesion, the lungs appearing perfectly normal. Brunner has reported a case of chronic pneumothorax in a man, aged 35, in whom the apex was fixed by a number of thin hollow adhesions with transparent membraneous walls. In two places the lung tissue was pulled out to a point, but the site of perforation could not be found. There was no evidence of tuberculosis.

We may now turn to the etiological explanation of the lesion as advanced by B. Fischer and his pupil J. Hayashi

on the basis of their findings in their above-mentioned three cases.

Fischer calls the disease: *"Spontanpneumothorax durch Ruptur von Spitzennarbenblasen — ein typisches Krankheitsbild"*, and he claims that all cases of spontaneous pneumothorax in apparently healthy individuals are due to a rupture of such scar-tissue vesicles.

These scar-tissue vesicles have been observed and described before Hayashi and Fischer called attention to them; and sometimes they are found on autopsy also in individuals who have never had spontaneous pneumothorax, as here the vesicles have kept from rupturing. Thus, Orth[1] has given a very accurate macroscopic description of these vesicles, but no particular significance was attached to them before Hayashi and Fischer published their studies. To these two investigators goes the credit for having demonstrated that these vesicles may give rise to spontaneous pneumothorax.

According to Fischer, the scar-tissue vesicles are formed through atrophy and inflation of the lung tissue in spots, resulting from the presence of some valve-like structure at the base of the vesicles. These "valves" develop as a result of small, localised, inflammatory processes with subsequent scar formation, which produces a constriction of the bronchioles with atrophy and deformity of the appertaining lung tissue as the scar is shrinking.

Hayashi is the first to make a thorough study of these vesicles. Even Orth has pointed out that there must be an obstruction to the outlet of the air from the vesicles, as it is very difficult or impossible to force the air out of them by pressure. On microscopic examination of the vesicles Hayashi found a valve-like structure at their base, on the order of the flap valve in an ordinary pump. This „valve" consists of deformed atrophic lung tissue. The valve allows the air to pass freely into the vesicle, but prevents it from passing out again. The effect of this will be, that the vesicle is gradually increasing in size. Finally, the wall

of the vesicle is so thin and atrophic in some spot that it ruptures on some occasion when the pressure is particularly increased (exertion), or it may rupture "of its own accord" under ordinary respiratory movements. So the valve mechanism is most important to the pathogenesis of the rupture as well as to the formation of the vesicle itself.

It is a peculiar fact that these vesicles in most cases are located at the apex of the lung. Fischer explains it as depending upon the circumstances that the apex is that part of the lung where scar tissue is located most frequently, and that the apices of the lungs are distended to a particularly marked degree on every increase of the intrathoracic pressure (e. g., coughing).

Of course, as also pointed out by Fischer, scar-tissue vesicles may be found in other parts of the lungs too. In case No. 21 of my material, where there was a partial pneumothorax in the lower part of the right side of the chest, the rupture must have taken place somewhere in this field. The same applies to the case reported by Koelensmid (p. 66.).

As to the origin of this scar tissue, neither Hayashi nor Fischer are able to offer any definite suggestion. Two of their cases (No. 4 and 6 in the survey above), which were examined very thoroughly, showed no sign of tuberculosis, neither macroscopic nor microscopic. Still, these authors consider a previous, very limited, tuberculous affection to be the most probable cause of the scar tissue; and in their third case (No. 3 in the survey above) the scattered scars and calcified foci in both lungs can hardly be interpreted as anything but the remnants of healed tuberculosis.

Own Investigations.

None of the 51 patients described in this study have died from their pneumothorax. But two of the patients, No. 12 and 21 have later died from other causes, and autopsy was made in one of these cases, No. 21.

On August 21, 1923, this patient (No. 21) had an attack of partial pneumothorax in the lower part of the right side of the chest, and he was treated for this condition in the hospital till October 2. After being perfectly well for some time, he had in the beginning of March, 1924, a severe attack of acute bronchopneumonia on the right side, which was subsequently complicated with empyema and pyopneumothorax, terminating fatally on April 4, 1924. *Autopsy,* at which I was not present myself, revealed no evidence whatever of macroscopic tuberculous changes in the lungs. The autopsy record says nothing about the possible presence of air vesicles.

In this case, then, the autopsy did not clear up the cause of the partial pneumothorax the patient was suffering from about half a year before he died. But *the autopsy showed that there were no demonstrable tuberculous changes in the lungs; and this is quite in keeping with the findings reported so far.*

A third patient in my material, No. 24, had subsequently a severe degree of progressive pulmonary tuberculosis, and she died from this lesion in connection with spontaneous pneumothorax (described in detail on p. 116.). *Autopsy* revealed a very thin-walled emphysematous vesicle (about the size of a goose-egg) on the surface of the lower lobe of the right lung.

To get some further information as to the etiology of the lession I have been looking for the scar-tissue vesicles described by Hayashi and Fischer in autopsies where such vesicles might possibly appear as an accidental finding in individuals who had never had spontaneous pneumothorax. In this search it was found that this kind of solitary vesicle formation is a rare phenomenon.

In the Pathological Institute of the Kommunehospital of Copenhagen, where ca. 700 autopsies are performed every year, such a valve vesicle was found only once in six months. Shortly after, another case of this vesicle formation (with typical scar-tissue vesicles) was found in the

Øresundshospital; and finally, six months later, a third case was found in the Kommunehospital. It would undoubtedly prove very interesting to investigate a fairly large number of such valve vesicles, but evidently it would take a long time, as these vesicles are far from being frequent. Still, the examination of the 3 cases mentioned has given some rather interesting results that throw some additional light on the etiology and pathogenesis of the lesion. I shall begin with a description of the first case from the Kommunehospital.

Case A.

The patient was a woman, 65 years old, who was admitted to Dep. II of the Kommunehospital on April 8, 1930, and died on April 11, 1930. Clinical diagnosis: Uræmia. Data from the case records: Past history of good health, apart from cholelithiasis. One year ago she was treated for chronic nephritis (increase of blood urea concentration). Several attacks of uræmia in the past month. Physical exam.: Hypertrophy of the heart and hypertension (enormous increase of blood urea). Auscultation of the lungs: No abnormalities.

Autopsy diagnosis: Nephritis chronica. Cystopyelitis. Hypertrophia cordis. Emphysema pulmonum. Cystis bullosa lob. medii pulm. dext. Focus calcificatus lob. inf. dext. Pleuritis chronica adhæsiva duplex. Cholecystectomia antea. Perihepatitis chron.

Pleuræ: Smooth and glistening, apart from some rather strong streaky adhesions over the apices and some delicate adhesions between the lobes of the right lung.

Lungs: Emphysematous, with hypostasis and oedema of the lower lobes. The lower lobe of the right lung is partially divided into two lobes, a smaller upper and a larger lower. At the posterior tip of the middle lobe is a clear vesicle the size of a large cherry, protruding freely from the surface of the lung; the pole of this vesicle is fixed to the opposite tip of the aforementioned upper part

Fig. 1.

Right lung with solitary emphysematous valve vesicle on the posterior tip of the middle lobe (case A). No other vesicles on the surface of the lung. A piece of cardboard is placed back of the vesicle.

Fig. 2.

Same emphysematous valve vesicle as shown in fig. 1, removed together with adjacent blocks of lung tissue. Natural size. (cf. fig. 3 and 4.).

of the lower lobe. No other bullous changes are observed. In the lower part of the lower lobe is a pea-sized calcified focus. The bronchial glands show no definite tuberculous changes.

Fig. 1 shows the site of this large solitary vesicle in the interlobar fissure between the middle and lower lobes. The lungs showed a moderate degree of emphysema, which could not be made out on the clinical examination of the patient.

The pea-sized calcified focus was located far down in the lower lobe of the right lung, at a considerable distance from the vesicle. Apart from this, a very thorough examination failed to reveal any macroscopically demonstrable cicatricial or tuberculous changes, neither in the vicinity of the vesicle, nor anywhere else in the lung.

Fig. 2 shows this vesicle removed together with adjacent parts of the lung. The vesicle is almost spherical, measuring ca. 2.2 cm. in diameter, arising from a broad base from the posterior tip of the middle lobe and attached at its opposite pole to the inferior lobe by a short adhesion.

The wall of this vesicle was thin as tissue-paper and transparent, with some branching blood vessels that were chiefly coming from the adhesion, while some smaller vessels came from the base of the vesicle. As the air could not be forced out of the vesicle by pressure, it must be regarded as a valve vesicle. Examination with a magnifying glass, after the vesicle was cut open, did not reveal any opening in its base.

Microscopic examination of the vesicle and its surroundings showed:

The lung tissue adjacent to the base of the vesicle shows marked emphysematous changes with distended thin-walled alveoli, disappearance of septa and dilatation of infundibula. There are no signs whatever of inflammation, scar formation or tuberculosis. The wall of the vesicle consists of an exceedingly thin layer of connective tissue, very poor in cells, covered on the inside with very flat cells.

The vesicle is unilocular, containing neither membranes nor septa. At the edge of the base the visceral pleura is seen to extend a short way out in the wall of the vesicle. Looking at the base of the vesicle, one finds a very thin membrane, separating the vesicle from the underlying emphysematous lung tissue. In this adjacent lung tissue, about at the middle of the base of the vesicle, is a branching bronchiole, of which one branch — measuring ca. 0.5 mm. in diameter — is proceding obliquely up towards the base of the vesicle, forming an angle of ca. 45^0 with the base of the vesicle.

In examining the serial sections of this area, one finds some sections wherein this bronchiole is shut off from the vesicle, and some sections (see fig. 3 and 4) in which this bronchiole appears to open out into the vesicle in such a way that the oblique course of the bronchiole gives rise to the formation of a tongue-like flap of emphysematous lung tissue. It is not certain whether this apparent inlet of the bronchiole into the vesicle is actually real, or whether the bronchiole may have been separated from the vesicle by a thin partition that has been broken in the handling of the specimen. Still, it is quite likely that the bronchiole is really communicating with the lumen of the vesicle in this particular spot. At any rate, this is the only bronchiole that is found in the vicinity of the base of the vesicle, and there can be no doubt that it is opening into the vesicle in such a way as to produce a sort of valvular mechanism, as no air could be forced out of the vesicle by pressure before it was cut open.

This case is thus presenting a very large valve vesicle, which is so thin-walled that it might readily have ruptured and given rise to spontaneous pneumothorax. The lung showed a moderate degree of emphysema, but not enough to be diagnosed on the clinical examination. So, if this

vesicle had ruptured, the condition would have been taken as a case of spontaneous pneumothorax in apparently healthy individuals.

That bullous vesicles of emphysema are not usually valve vesicles, I have ascertained by examining some specimens from patients whose lungs showed multiple bullous emphysema vesicles.

Thus there was the case of a woman, aged 76, who died with uræmia on June 16, 1930, in the Medical Department of the Øresundshospital. No emphysema of the lungs could be made out on clinical examination, but the autopsy showed numerous bullous vesicles of emphysema scattered over the surface of the lungs. The right apex was the site of a group of emphysematous vesicles as large as beans (fig. 5).

On examination of serial sections from these vesicles, most of the sections showed free communications between the three large emphysematous vesicles and the underlying lung tissue, so that these vesicles could not at all be designated as valve vesicles. By a peculiar stroke of chance, in addition to the three large emphysematous vesicles, the apex of this lung showed also a scar-tissue vesicle with a base of dense cicatricial tissue. There might possibly have been some sort of a valve in connection with this vesicle, but the vesicle is so thick-walled that it cannot be designated as ready to burst.

It is very seldom that patients with bullous emphysema of the lungs have spontaneous pneumothorax, and this is probably due to the fact that these vesicles are rarely valve vesicles and as a rule are communicating freely with the underlying lung tissue.

In case A, however, we have an illustration of the fact that large solitary valve vesicles *can* develop in lungs in which there are only emphysematous changes and no scar tissue whatever; and there can be no doubt that, by rupturing, such vesicles may give rise to spontaneous pneumothorax. If the emphysema of the lungs in such a case can not

be diagnosed clinically, one will naturally enter the case under the term: Spontaneous pneumothorax in apparently healthy individuals.

I shall then describe the second case, which, in contrast with the first, showed typical scar-tissue vesicles.

Case B.

The patient was a woman, aged 82, who was admitted to the Medical Department of the Øresundshospital on April 28, 1930, and died on May 4, 1930. Data from the case record: Pleurisy at the age of 5 years, otherwise past history of good health until 7 *years ago* when she was admitted to this hospital for pulmonary tuberculosis with hæmoptysis. The patient was under hospital treatment from February 9 to June 22, 1923. At this time, the apex of the right lung was affected. The expectorate was examined 8 times, showing once + T. B. On her discharge, the patient returned to her home, and she has been feeling well the past 7 years. She is now (April 28, 1930) admitted to the hospital with symptoms of acute enteritis. *Auscultation of the lungs* shows some dullness of the right apex. Expectorate: ÷ T. B. She is gradually getting worse, with increasing cloudiness of her mind and rise of temperature; and she dies on May 4.

Autopsy diagnosis: Pneumoniæ lobulares confluentes lobi inf. dext. med. gr. Cicatrices et foci calcificati apicis pulm. d. Pleuritis adhæsiva fibrosa apicis pulm. d. levi gradu. Emphysema pulm. Arteriosclerosis universalis. Hypertrophia cordis. Enteritis follicularis. Obesitas. Atrophia organorum med. gr. inprimis renum. Cystitis chronica. Fibromyoma uteri.

Lungs: The right apex is adhering to the chest wall by strands of fibrous tissue, and it contains some darkly pigmented scars (up to bean-size), forming depressions of the surface. At the tip of the apex are two bean-sized

Fig. 5.

Microphotograph of apex of a lung from a patient with bullous emphysema of the lungs. Magnif. × 3.

To the right, above, are 3 large emphysematous vesicles which are *not* valve vesicles. At the left is a small area of dense cicatricial tissue. Above this, and to the left, is a scar-tissue vesicle that possibly may be a valve vesicle. Here, then, in the *same* apex of the lung are *both* a group of ordinary emphysematous vesicles *and* a solitary scar-tissue vesicle.

Fig. 6.

Apex of a lung with scar-tissue vesicles, retractions of the surface and adhesions (case B). 4/5 natural size.

Above the mark ×, in the middle of the picture, is one of the large scar-tissue vesicles, immediately to the right of a deep cicatricial retraction. Medially to the other mark ×, protruding from the surface of the lung is the other large scar-tissue vesicle. In the area between the two large vesicles, the surface of the lung is arching a little from the presence of several small vesicles. (cf. fig. 7 and 8.).

emphysematous bullæ. Inside the scars and elsewhere in the lungs are several calcified foci, the size of millet seeds. Some of the bronchial glands are moderately enlarged, several are anthracotic; they show no sign of fresh tuberculosis, and they contain no caseous or calcified foci. In addition, the lungs show a moderate degree of general emphysema and areas of bronchopneumonia in the lower right lobe.

The picture of the right apex (fig. 6) shows the above mentioned adhesions and cicatricial depressions, surrounding the two bean-sized protruding vesicles. The most prominent of the vesicles, which is seen in the middle of the picture, is located just alongside a deep cicatricial retraction of the surface, produced by a large wedge-shaped scar with its base on the surface of the lung and extending a couple of centimeters down in the lung tissue. This scar, which consists of dense fibrous tissue, envelops a calcified focus, the size of a millet seed. In addition, there are other patches of scar tissue, some in the form of a localised thickening of the pleura, some as streaks of connective tissue, extending down in the lung. Between the two large vesicles is an area where the surface is nodular through the presence of a group of smaller vesicles. On section these small vesicles (ranging from millet-seed to pea size) are found to be crowded together under the surface. Some of them are multilocular, with a system of partitions and fine cords. These small vesicles are also surrounded by scar tissue, and they are most likely to be interpreted as representing an early stage of the large scar-tissue vesicles, even though their structure is more suggestive of bullous emphysema.

Microscopic examination:

The surroundings of the vesicles are composed of scar tissue with intermediate patches of lung tissue showing marked emphysematous changes. The cicatricial tissue consists of dense fibrillary connective tissue with large

deposits of anthracotic pigment. No sign of fresh tuberculosis. Here and there in the connective tissue are some scattered — often peribronchial — accumulations of lymphocytes, but tubercle formation is not observed in any of the sections from this part of the lung.

The wall of the vesicles varies somewhat in thickness in different parts of the same vesicle; it consists of fibrillary connective tissue with a few branching blood vessels.

Examination of a number of sections from the largest vesicle shows, as illustrated in the microphotograph fig. 7, that the vesicle is resting on a base of massive scar tissue in which there are no remnants of the original alveolar structures. The scar tissue contains some branching blood vessels, and a little distance below the vesicle is a small bronchus, but no direct communication is found between this and the vesicle. In some sections one finds in the periphery of the base, immediately beneath the vesicle, a small area containing some flattened, hollow spaces. The air-filled vesicle is undoubtedly in some communication with these hollow spaces, but, unfortunately, this communication cannot be demonstrated in any of the sections examined.

The other large vesicle is a little broader and less protruding. The base of this vesicle is formed in part by markedly emphysematous lung tissue, and partly by greater or smaller streaks of connective tissue. On the microphotograph fig. 8, one will notice an area where these streaks of connective tissue are rather thin, while the base of the vesicle is mostly composed of a system of membranes enclosing flattened hollow spaces of varying size which are presumably communicating with the lumen of the vesicle in such a way as to produce the function of a valve mechanism. The structures of this tissue are so entangled, however, that it is impossible to have any clear idea as to how this valve mechanism is built. But, as is evident from fig. 8, there can hardly be any doubt that at this place the membranes form some kind of a valve.

Fig. 7.

Microphotograph of the middle scar-tissue vesicle in fig. 6 (case B). Magnif. × 8.

The base of the vesicle consists of perfectly massive scar tissue, without any suggestion of normal lung tissue. A bronchus is seen in the scar tissue below the middle of the vesicle.

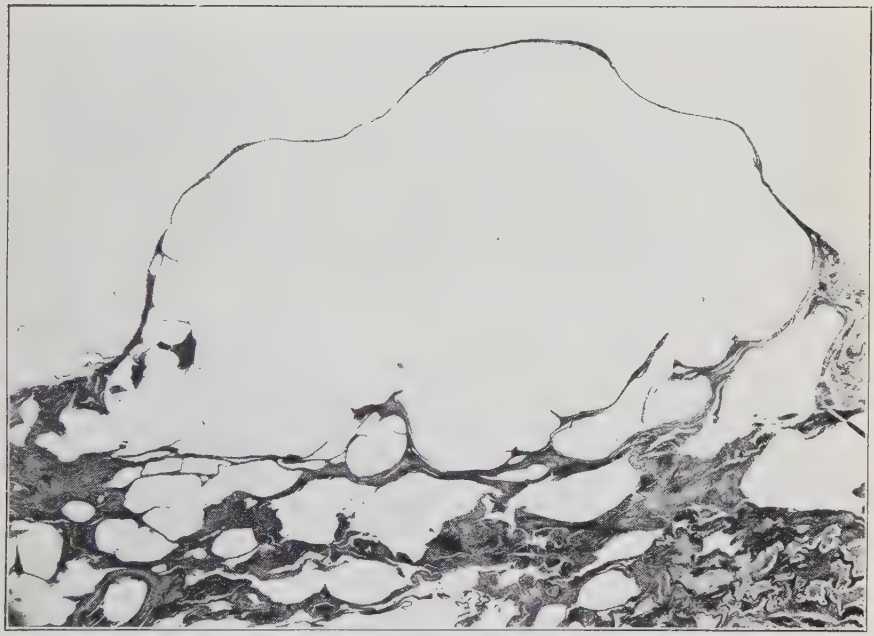

Fig. 8.

Microphotograph of the other large scar-tissue vesicle from case B. (cf. fig. 6.). Magnif. × 8.

The base of this vesicle is formed in part by lung tissue with marked emphysematous changes and partly by streaks of connective tissue, which forms a system of membranes enclosing some flattened hollow spaces.

In this case, then, we find valve vesicles that are quite different in origin and structure from the valve vesicle in case A. Here, in case B, the apex of the lung shows typical multiple scar-tissue vesicles of quite the same character as encountered in those cases where Hayashi and Fischer found them to be the underlying cause of spontaneous pneumothorax in apparently healthy persons.

Case B is of particular interest by this, that 7 years previously the patient had had a mild degree of tuberculosis in the same apex where now the scar-tissue vesicles were found. Hayashi and Fischer were not able to demonstrate that the scar tissue in their cases (No. 4 and 6 in the survey on p. 21.) was due to tuberculosis, even though they considered this the most probable. But here in case B we have an instance of valve vesicles being formed in the apex of the lung as a result of a healed tuberculous process.

In chronic pulmonary tuberculosis where productive and exudative forms of inflammation are encountered along with fibrous scar formation, one may also find scar-tissue vesicles on the surface of the lungs (Huebschmann). It is therefore reasonable to assume that such scar-tissue vesicles may give rise to spontaneous pneumothorax in consumptives too. But this question falls outside the scope of the studies here presented.

The next case showed a solitary scar-tissue vesicle in the apex of the lung, and here the valve could be demonstrated on microscopic examination.

Case C.

The patient was a man, 24 years old, who was admitted to the Blegdamshospital on October 30, 1930. Clinical diagnosis: Gangræna pulm. sin. Pyopneumothorax sin. Diabetes mellitus. On November 15, the patient was transferred to Dep. V of the Kommunehospital. Here, on November 17,

rib resection was performed on the left side, under local anæsthesia. The patient died on the November 26.

Autopsy diagnosis: Diabetes mellitus. Bronchitis chron. Bronchopneumonia pulm. utr. Bronchiectasia pulm. utr. Abscessus pulm. sin. Gangræna pulm. sin. Empyema pleuræ sin.

Lungs: Right lung rather voluminous, somewhat rigid. On the cut surface which is marmorated greyish-red in colour, rather moist, are some irregular patches, light greyish-red in colour and not containing any air. These areas are more or less confluent, alternating with hyperæmic patches that contain some air. There is a purulent material in the small bronchi. *At the apex is a ring-formed thickening of the pleura, and from the centre of this, an air-filled vesicle is protruding.*

Left lung is somewhat collapsed, but there is still some air in the tissue. In the middle of the upper lobe is an abscess cavity (the size of a hazel-nut) and between this and the lateral surface — a little further down — is another abscess cavity (almost as large as a walnut) with ragged edges and perforating through the pleural surface. The small bronchi are plugged with pus and surrounded by peribronchial infiltrations.

Bronchi: The large bronchi are somewhat distended, showing marked injection of the mucous membrane. The small bronchi of both lungs are distended; the mucous membrane is congested and somewhat swollen.

Pleuræ: Right pleura shows no definite abnormality apart from the aforementioned thickening with the air vesicle at the apex.

Left pleura is markedly thickened throughout and covered with a greyish-yellow fibrinous exudate. At the base of the left pleural cavity is an encapsulated empyema, about the size of a large walnut.

The bronchial glands are swollen, but they show no abscess formations or tuberculous processes.

Heart and aorta appear to be normal.

(The autopsy permit covers only the organs in the chest.)

Further examination of the *apex of the right lung:*

At the very top of the apex is a protruding air vesicle (the size of a large pea), encircled by a thickening of the pleura. There are no retractions of the surface and no adhesions; there are no other vesicles but this one. The vesicle is whitish in colour, non-transparent and rather thick-walled. No blood vessels can be seen in the wall of the vesicle. On compression of the surrounding lung tissue the vesicle is further distended, but diminishes somewhat again as soon as the pressure on the lung tissue is discontinued.

Microscopic examination:

The vesicle is examined in serial sections, every third section being stained (a total of 157 stained sections).

The vesicle is almost spherical, arching above the pleural surface of the lung. The wall of the vesicle consists of fibrillary connective tissue, without any signs of inflammatory processes; it measures ca. 0.2 mm. in thickness, which corresponds to the normal thickness of the pleura (0.13—0.24 mm., according to Zahn).

At the junction between the wall of the vesicle and the pleural surface of the lung is a small ledge of subpleural connective tissue, extending from the pleural surface about 5 mm. down into normal non-emphysematous lung tissue. In most of the sections this scar tissue appears as an almost square mass of connective tissue, measuring ca. 5 mm. in height and width. On comparison of the various sections, it is evident that this connective tissue structure encircles the vesicle like a ring in horseshoe form, but in such a way that the greatest part of the scar tissue is located by the side of the vesicle.

The peripheral sections — i. e., the first and the last sections of the series — show that the base of the vesicle here is separated from the underlying lung tissue by a

membrane or a stem of connective tissue. But in the sections from the middle of the specimen, where the vesicle is cut in its widest diameter, the scar tissue forms only about one half of the base of the vesicle (in fig. 9 this is seen to the left of the mark x). In these sections the other half of the base of the vesicle consists of partly emphysematous lung tissue with irregular and confluent alveoli, interspersed with a few streaks of connective tissue. This altered lung tissue is seen in the lower right corner of fig. 9. The alveolar spaces are filled with an exudate rich in cells.

The scar tissue itself is not composed of perfectly compact connective tissue, but consists of streaks of fibrillary connective tissue enclosing larger and smaller spaces, which have resulted from alteration of the original lung tissue. The scar tissue is infiltrated with a rather large number of lymphocytes, partly scattered in the connective tissue, partly accumulated in small heaps. None of the sections show any evidence of tubercle formation. Anthracotic pigment is seen here and there in the scar tissue.

Several sections from the middle of the vesicle show the outlet of a small bronchus into the vesicle, establishing a communication between the vesicle and the underlying segment of the lung. Fig. 9 shows this little bronchus (0.1—0.2 mm. in diameter) entering the vesicle obliquely, forming an angle of ca. 30^0 with the base of the vesicle.

It is seen very plainly that the tissue between this bronchiole and the vesicle is here forming a tongue-shaped flap that must act as a valve. This flap will let the air pass into the vesicle, but it will shut off the passage against outgoing air. Fig. 10, a microphotograph (magnif. $\times$ 50), shows that the tongue-shaped flap consists of dense fibrillary connective tissue, enclosing a single large tissue space. So the flap is formed of scar tissue.

There was no other demonstrable communication between the vesicle and the underlying lung tissue.

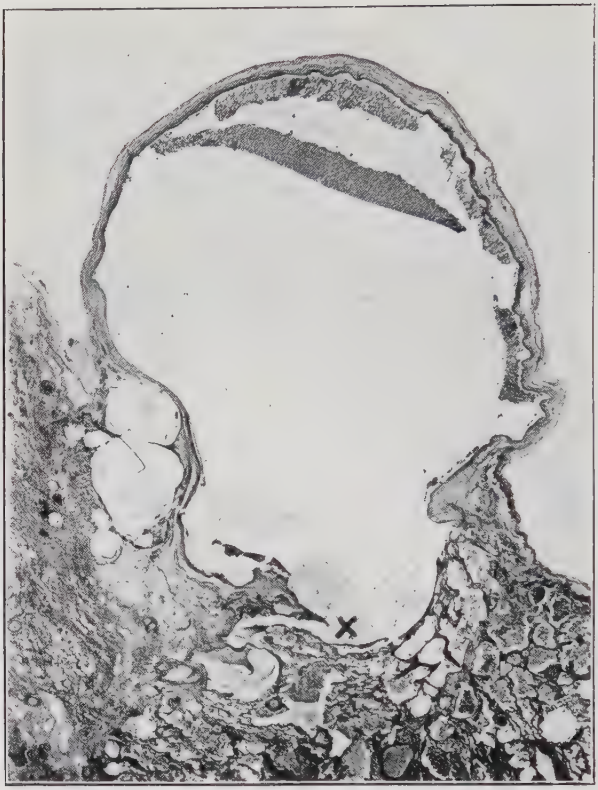

Fig. 9.
Scar-tissue vesicle in case C. Microphotograph. Magnif. × 10. The mark × is placed at the outlet of a small bronchus into the vesicle.

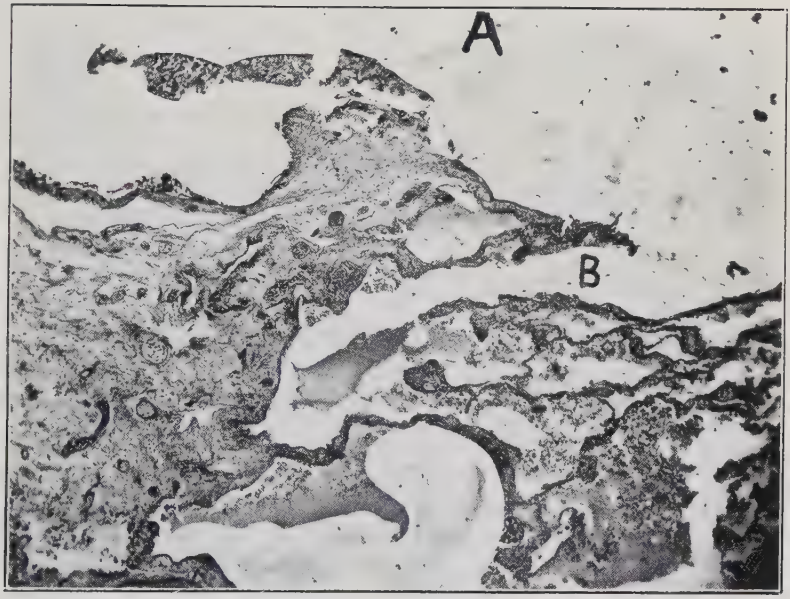

Fig. 10.

Scar-tissue vesicle in case C. Microphotograph of section from the same field as shown in fig. 9. Manif. × 50.

The picture shows the outlet of a small branch of a bronchus into the vesicle.

A: Cavity of vesicle. B: Lumen of bronchus.

Here, then, we have the case of a young man whose lungs show no signs of emphysema or tuberculosis, whereas one lung presents a solitary scar-tissue vesicle at its apex. The microscopic examination has shown that at the base of this vesicle a small branch of a bronchus enters the vesicle obliquely, in such a way that the tissue here forms a tongue-shaped flap which can undoubtedly act as a valve. So there can hardly be any doubt that this is a valve vesicle. Moreover, the anatomical relations of this vesicle are such as to make it probable that, if the patient had lived, this vesicle might have developed further until, eventually, it would rupture and give rise to a spontaneous pneumothorax.

Comparing this vesicle with the vesicle in case A, one finds that they show exactly the same mechanism of bronchial inlet. In both cases a small branch of a bronchus enters the base of the vesicle in such a way that the tissue here forms a tongue-like flap valve. The only difference between the two cases is, that in case A the valve contains no scar tissue but consists of emphysematous lung tissue, whereas in case C the valve is made of scar tissue.

Both these vesicles are valve vesicles, but in case A we have an emphysematous valve vesicle, in case C a cicatricial valve vesicle.

There can hardly be any doubt that these two forms of valve vesicles by rupturing may both give rise to the clinical picture of spontaneous pneumothorax in apparently healthy persons.

These anatomical examinations show then, that there are two different forms of valve vesicles. Both forms have a valve mechanism at the base of the vesicle. In one of these (case A), the valve is formed entirely through local emphysematous changes in the lung tissue; in the other

type the valve mechanism results from shrinkage of scar tissue (cases B and C).

Hayashi has also described the occurrence of valve vesicles without any scar tissue in the valve mechanism. He has examined 4 instances of solitary air vesicles on the pleural surface of the lungs, that were found accidentally on autopsy. In two of these cases the bullous emphysema vesicle was formed in direct connection with a fibrous tuberculous focus, but in one case the localised consolidation of the lung tissue was too insignificant and too far away from the emphysema vesicle to suggest any connection between the two processes; and in his last case, a bean-sized air vesicle was found on the anterior margin of the right lung, where the microscopic examination showed moderate simple emphysematous changes without any scar tissue.

When, later, Fischer calls attention to the presence of shrinking scar tissue as the main factor of the formation of valve vesicles, he is undoubtedly in the right, as the presence of a shrinking scar tissue gives the best possibilities for the development of a valve mechanism.

It should be emphasized, however, as shown in my case A, that even very large valve vesicles — ready to rupture — may be formed without the presence of any scar tissue whatever.

Here it may be mentioned that the literature contains 3 reports on familiary occurrence of spontaneous pneumothorax in apparently healthy persons (Erik E. Faber, Kusan, Atwood). In view of the infrequency of this lesion, it is not very likely that the occurrence of several cases within a family would be merely accidental. It is more probable that these patients have some congenital disposition to the formation of valve vesicles, but none of these cases has been examined post mortem.

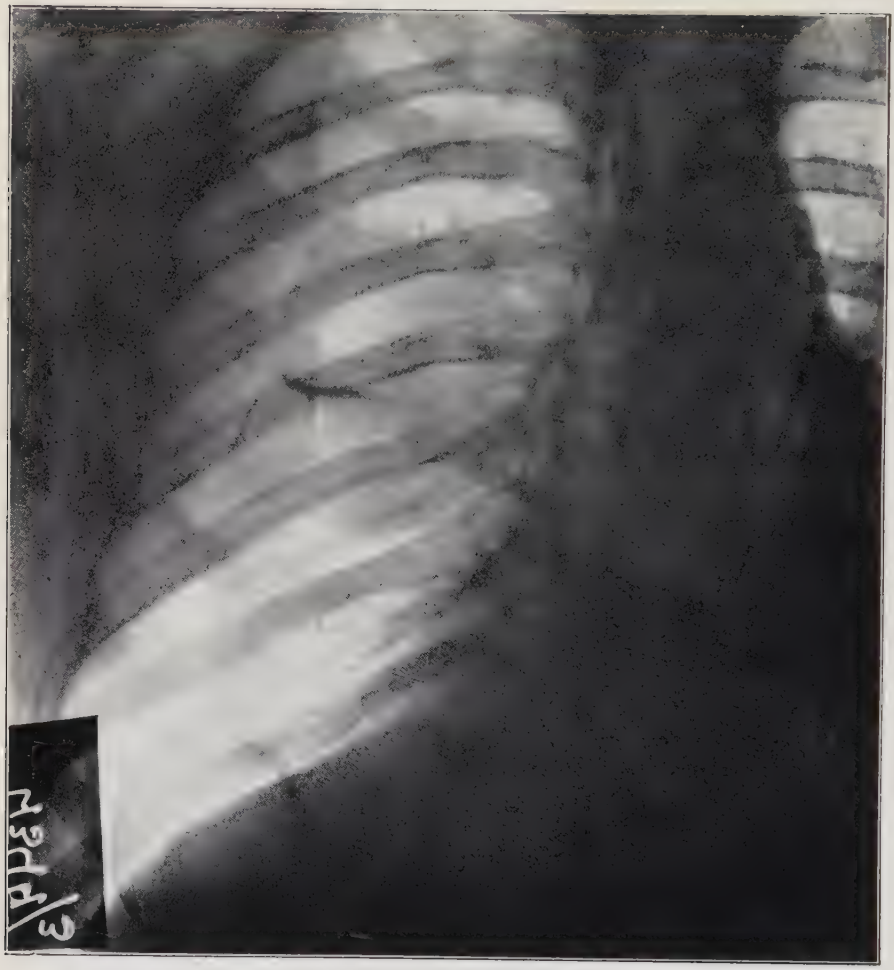

Fig. 11.

Pt. No. 42 *c*. Right lung, half-way expanded, with distinct contours of the individual lobes. At the bottom of the pleural cavity and over to the side (just inside the mark with the registration number of the plate) is a large ovoid contour (shadow-producing vesicle, resting on the diaphragm).

Before concluding the chapter on the etiology of spontaneous pneumothorax in apparently healthy persons, one more question arises, namely:

When these attacks of spontaneous pneumothorax are due to the rupture of a valve vesicle, is it not possible, then, that such a vesicle may occasionally show on the X-ray plates?

In general, one certainly cannot expect to see anything whatever of the valve vesicles on the X-ray plates, because the vesicles are extremely thin-walled, and they collapse when they rupture. It is quite true that a Roentgenogram of a removed lung with bullous emphysema will show the fine outlines of the blebs, but no such structures may be demonstrated on X-ray examination of the lungs in situ.

In my material, however, there is one case in which the valve vesicle can be made out on the X-ray plates, and this is due to particularly favourable conditions.

Within 4 years, this patient — No. 42 — had 3 attacks of spontaneous pneumothorax on the right side. During his third attack (Case Record No. 42, *c*) the X-ray plates showed a contour of the size and form as a hen's egg, situated on the surface of the lower right lobe. This picture naturally led to a thorough examination of the previous Roentgenograms of the patient, with this result (see fig. 11):

Even the plates from the first attack, in 1927, show — at the foot of the lateral margin of the lower lobe of the right lung — a delicate outline of a protuberance on the lung border, about the size of a walnut.

On the last plates, from the attack in 1931, one finds, on the very same site, a configuration — of size and form as a hen's egg — protruding from the lower lung border laterally out in the pneumothorax cavity (fig. 11).

This contour can hardly be interpreted as anything but a large subpleural vesicle at the margin of the lung, with a rather thick wall consisting of fibrous tissue (otherwise it would not be so conspicuous) and possibly containing

some deposits of lime salts. The continuity of this contour is broken only over a small area of its upper lateral section. Thus, it is reasonable to assume that in this section the wall of the vesicle has not been as thick as elsewhere, so that it does not show on the plate, and that the rupture of the vesicle has taken place in this thin section of the wall.

It is most probable, then, that these X-ray plates do actually show the valve vesicle which, by rupturing, has given rise to the three attacks of pneumothorax in this patient.

This case is quite unique, as the X-ray plates from the attack in 1931 furnish the only instance in my material where, by Roentgenography, it has been possible to demonstrate the presence of a vesicle on the surface of the lungs.

That in this case the third attack of spontaneous pneumothorax is brought about by the rupture of a valve vesicle as large as a hen's egg is not improbable, even though the valve vesicles giving rise to spontaneous pneumothorax are as a rule much smaller (from very tiny up to nut-sized). In this connection, I may mention that the autopsy on Pt. No. 24 (see p. 117.), who had had 4 attacks of pneumothorax, revealed an air vesicle as large as a goose-egg that was also protruding from the lower margin of the lower lobe of the right lung — that is, at the same site as the large vesicle in Pt. No. 42.

In summing up the findings concerning the etiology of spontaneous pneumothorax in apparently healthy persons, the result is:

The disease is due to rupture of a valve vesicle on the surface of the lungs. There are two forms of valve vesicles. One form, the *scar-tissue vesicle,* develops especially in the apices of the lungs, next to scars that may result from healed tuberculosis. These scar-tissue vesicles are often

multiple; they are probably the most frequent form of valve vesicles.

The other form, the *emphysematous valve vesicle,* develops through local emphysematous changes without scar tissue. The emphysematous valve vesicles may occur as large solitary vesicles on the lung borders.

But, of course, this does not by any means settle the clinical side of the problem, for now arises the question:

When, presumably, several of these patients have tuberculous scar tissue in the apices of the lungs, does this not really imply a latent form of tuberculosis, and are not these patients to be treated as mild cases of pulmonary tuberculosis?

Some authors have reported instances of spontaneous pneumothorax in apparently healthy persons where there has been reason to assume that tuberculosis may have been the underlying condition. A few of these patients have given a past history of a mild degree of tuberculosis with recovery (e. g., Dahlstedt & Haeger, Case No. 2) or good health after hæmoptysis (Sale's case). In other cases, latent tuberculous foci have been demonstrated on X-ray examination after the lungs have expanded (Coghlan, Rieder, v. Teubern, Deetjen, George A. Allan).

In other words: Can one be sure that this scar tissue, which some of these patients must be assumed to have in the apices of their lungs, does not contain the germ of a subsequent serious case of pulmonary tuberculosis? And how is the clinical picture of this lesion under discussion to be defined?

These are the questions — of such a decisive importance to the physician — which I have tried to elucidate through clinical investigations.

DEFINITION AND DIFFERENTIATION

The spontaneous pneumothorax in apparently healthy persons has to be distinguished, on one side, from pneumothorax in patients with emphysema of the lungs, and, on the other side, from pneumothorax in patients with pulmonary tuberculosis.

Galliard[1]) has emphasized that distinction should be made between

1) pneumothorax in patients with pronounced emphysema (*Pneumothorax des grands emphysémateux*), and
2) pneumothorax in patients with latent emphysema (*Pneumothorax des petits emphysémateux*).

In the last group, Galliard included patients with pneumothorax of non-demonstrable origin, as in such cases he took the cause to be a rupture of a small non-demonstrable emphysematous vesicle.

Galliard and, later, Fischer as well as Enneking emphasize that the course and prognosis of spontaneous pneumothorax are considerably more serious in emphysematous patients, than in persons who are otherwise healthy. In spontaneous pneumothorax the prognosis is chiefly dependent upon the condition of the other lung and the heart; and this lesion is therefore always a serious complication in patients with emphysema, especially in the severe degrees. This clinically different lesion is then to be distinguished from spontaneous pneumothorax in the apparently healthy; and this will not be difficult, as a marked degree of emphysema is easy to recognise.

The other main point in the delimitation of the clinical picture of the lesion is its differentiation from spontaneous pneumothorax in tuberculosis.

It is to be kept in mind that there is a wide gap between the estimation of an autopsy finding and judging a case in the clinic. In the clinic, one is facing far greater difficulties than at an autopsy, and the question is then, whether by the clinical diagnostics one may be able to rubricate a case with such a certainty that one may reckon that the clinical features of the case in question really correspond to the aforementioned etiological factor: the rupture of a valve vesicle.

In attempting to define the clinical features of the lesion one must aim to ensure that cases with an underlying condition of latent tuberculosis are left out, so that the term finds application only to cases representing something entirely different. For it is obvious that if it is not practicable clinically to differentiate between the cases arising from the rupture of a valve vesicle and cases due to some form of tuberculosis which progresses later on, it is not justified at all to maintain the term spontaneous pneumothorax in the apparently healthy.

If so, it would be best to give up this clinical picture as a nosographic unity, and it would be proper to consider all such cases suspicious as to pulmonary tuberculosis, and treat them accordingly.

So the justification of maintaining this clinical entity as a nosographic unity stands and falls with the prognosis.

So far, there have been but very few and defective data available as to the subsequent condition of these patients, so that one has naturally been wavering very much in judging these cases.

My principal aim with the studies here presented has therefore been through systematic after-examinations to give the wanting basis for the estimation of the prognosis in these cases.

As will be shown later, the result has been that in 49 after-examined patients only one has pulmonary tuberculosis subsequent to spontaneous pneumothorax. This patient was exposed to tubercular infection in her home,

so that it would hardly be proper in this case to associate the later tuberculosis with her attack of spontaneous pneumothorax, especially as the two lesions were located in different lungs. None of the other 48 patients have shown signs of pulmonary tuberculosis — yet most of them have been under observation a number of years.

These after-examinations have thus established how uniform and benign is the prognosis.

I think, therefore, that it is possible to maintain a practically serviceable, clinical, picture of the disease with typical course and prognosis, and that my differentiation of its clinical features has proved to be of practical value. The main point is that, according to my findings, it is warrantable in typical cases to reckon that these patients do *not* have pulmonary tuberculosis later on. This is the chief importance attached to the establishment of this clinical unity.

In order not to blur the features of this lesion and make uncertain its prognosis, I think the term of spontaneous pneumothorax in apparently healthy persons should *not* cover any cases in which the patients previously had been suffering from pulmonary tuberculosis.

Still, if one is meeting with an otherwise typical case of spontaneous pneumothorax in a patient who gives a past history of a very mild attack of pulmonary tuberculosis many years before, and *where this tuberculosis has been perfectly healed clinically and non-demonstrable for a number of years,* I find it justified to enter such a case under the heading: spontaneous pneumothorax in apparently healthy persons. In such a case, the spontaneous pneumothorax will most probably be due to the rupture of a scar-tissue vesicle, and the anatomical basis of the lesion will resemble the findings described above in the anatomical examination of the lungs in case B.

But, as will be mentioned later, none of my 51 patients

with spontaneous pneumothorax gives a history of pulmonary tuberculosis in the past.

Patients with typical spontaneous pneumothorax who give a past history of scrofulosis or pleurisy a good many years before and perfect health in the intervening years may appropriately be included under the term of spontaneous pneumothorax in the apparently healthy. My material covers 6 cases of this kind.

In the physical examination, of course, neither the auscultation nor the examination of the expectorate must show any sign of tuberculosis if the patient is to be included in this group. As to the auscultation, however, one should not attach too much importance to a very slight dullness or a faint bronchial note over the apices, for, indeed, such minor changes are frequently found without their meaning anything.

Next, it is to be emphasized strongly that the X-ray plate must show normal conditions. By this is meant that the Roentgenogram must not show any abnormalities beside the ones so frequently encountered in perfectly healthy individuals — calcifications of primary affections and hilus glands and minimal spots in the apices. This decisive X-ray examination cannot be made till the affected lung is again fully expanded, as sometimes a latent infiltration will not be revealed by Roentgenography unless the lung is expanded (Coghlan, Rieder, v. Teubern, Deetjen).

In patient No. 35 the other lung showed scattered calcifications, subsequent to pleurisy in childhood (see fig. 13, 14, 15). In case No. 39 the other apex showed a distinct fibrous scar; but as this kept unchanged for four years, and as the patient had never presented any symptoms of tuberculosis, it is reasonable to consider this a scar of long standing.

As to the course of this lesion, it is very important that the clinical picture covers only non-febrile cases which are not complicated with pleurisy.

As pointed out later, in the clinical section, a transitory

rise of the temperature in the first week of illness is of no conclusive significance. It is observed sometimes, without meaning anything.

In 2 of my cases friction sounds were heard in the first days of the illness, and then they disappeared; and I do not think that any importance should be attached to their transitory presence. One of these patients, No. 1 gave a negative Pirquet reaction, and he has been well since his attack of spontaneous pneumothorax; the after-examination, $18^3/_4$ years later, did not reveal anything suggestive of pulmonary tuberculosis. The other patient, No. 21, died from an other disease, $1/_2$ year after his attack of spontaneous pneumothorax. Autopsy revealed no macroscopic sign of tuberculosis in the lungs.

On the other hand, those cases of spontaneous pneumothorax where the presence of a pleural exudate can be demonstrated on auscultation, are not to be entered under the term: spontaneous pneumothorax in apparently healthy persons. For such cases are suspicious of an active tuberculous pleurisy. Most of these cases will also be accompaied by a rise of the temperature (cf. the aforementioned case of Letulle (p. 19.)).

Going through the literature, one will find a non-inconsiderable number of cases, wherein there has been a small exudate that could be demonstrated on percussion as a small area of dullness. Most of these cases in which there has been a small exudate, have recovered rapidly, but data are wanting as to how these patients have been getting along later on. I think that such cases are not to be included under the term: spontaneous pneumothorax in apparently healthy persons. In dealing with such cases, I think, one ought to insist upon the same regimen as in exudative tuberculous pleurisy and see that the patients go through an after-treatment in a tuberculosis sanatorium.

On the other hand, I think that the term spontaneous pneumothorax in apparently healthy individuals may very well be applied to cases in which the X-ray plate shows an

insignificant exudate in the phrenocostal groove, when it is so small that it cannot be demonstrated on auscultation.

My material includes 10 cases where there has been a small, clinically non-demonstrable, exudate which showed on the X-ray plate (cf. fig. 23). In 8 of these cases (No. 6, 13, 20, 33, 35, 38 *a*, 39, 41 *b*) there has been only a very small shadow of fluid in the phrenocostal groove. None of these patients has subsequently shown any signs of pulmonary tuberculosis. One of them (No. 33) gave a negative Pirquet reaction. One patient (No. 41) had no pleural effusion during the first attack of pneumothorax but showed a shadow of a minimal exudate in the phrenocostal groove during the second attack.

One of my cases, No. 25, may perhaps leave a little doubt as to whether it properly belongs to this group of cases. On admission to the hospital, 2 weeks after the onset of the illness, the patient showed left-sided pneumothorax without the slightest exudate. 1 week later, however, the X-ray examination revealed a small amount of fluid in the left pleural cavity, reaching to costa V on the anterior surface. The patient was altogether afebrile. On after-examination it is found that he has been perfectly well during the past 4 years, since his attack of pneumothorax, and there is no sign of tuberculosis. In another case (No. 5) the X-ray plate showed a little more pleural exudate, than did the aforementioned 8 cases, but not enough to be made out on auscultation, and the fluid disappeared rapidly. The patient has been well since ($14^3/_4$ years).

Finally it is to be kept in mind that such a small amount of fluid may in some cases be blood — as in case No. 36 (see fig. 21 and 22).

In other words: When one has to decide whether a case of spontaneous pneumothorax is a "pure" case of the lesion here in question, one has to go through with the same examinations and considerations as when, in another case,

one has to decide whether the patient has a latent tuberculosis or not.

In the past, undoubtedly, there has been a tendency to overestimate the difficulties of this decision and to attach too much importance to stethoscopical subtleties. Really, this decision is not particularly difficult to an experienced examiner with all the modern adjuvants to his disposal; nor will it as a rule be difficult to decide whether a patient with this lesion is clinically in good health. Doubtful cases will certainly be rare.

The conclusion is then: The clinical entity "spontaneous pneumothorax in the apparently healthy" covers every case of spontaneous pneumothorax that appears without any demonstrable cause in healthy persons, in whom tuberculosis cannot be demonstrated — neither by examination of the sputum, by auscultation, nor by X-ray examination — and in whom the lesion is not accompanied by fever or by pleural effusion. This category comprises also the cases in which there is a slight rise of the temperature in the first week of illness, and cases in which the X-ray examination reveals the presence of an insignificant exudate, too small to be made out on auscultation.

That it is warrantable to formulate this clinical entity with the given definition, is evident from the good prognosis of such cases as established by the material here presented.

FREQUENCY, OCCURRENCE AND LOCALISATION

FREQUENCY

Spontaneous pneumothorax in the apparently healthy is a comparatively rare disease.

The material of 51 patients here presented comprises all the cases it has been possible for me to gather on application to the hospitals in Copenhagen, to ca. 50 hospitals in the country, and to some practising physicians who, I knew, had been treating such cases. The period during which these cases have appeared, covers more than 20 years. In view of the fact that this material has thus been gathered from the entire Denmark, the lesion appears to be very rare. Still, in later years — particularly the past 5 years — the disease has been observed more frequently, and this will probably mean, that the lesion is not quite so rare as thought before, and that the correct diagnosis is now being made more frequently than before.

On the other hand, I can fully confirm that spontaneous pneumothorax in patients with pronounced chronic emphysema of the lungs must be regarded as a truly rare disease, for only one single case of this kind was observed in all the above-mentioned hospitals.

OCCURRENCE

It has been emphasized already by Hall and by Galliard that *this disease occurs most frequently in younger persons and more often in men than in women;* and my material is quite in agreement with this view.

In my 51 patients there are:

36 men,
15 women.

The age distribution of these cases is as follows:

0—10	years:	1 case.
10—20	„	5 cases.
20—30	„	22 „
30—40	„	14 „
40—50	„	9 „

The oldest of my patients was 49 years old. The youngest patient, No. 48, was only 5 years old, but it is very well possible that in this case the etiology of the lesion may have differed from that of the other cases (see p. 106.).

In case No. 32 the condition of chronic pneumothorax was not ascertained till the patient was 19 years old, although the lesion probably set in when the patient was only 5—6 years old.

A priori, one might imagine that this form of spontaneous pneumothorax would occur by far the most frequently in persons doing hard labour. But this is not the case.

Distributed after occupation, the 50 adults of my material fall as follows:

Heavy physical work (Blacksmith, farmer, baker, etc.)	13
Household work (Housewife, servant)	10
Less exhaustive, physical, work (Electrician, shoemaker, etc.)	8
Shop clerks, functionaries, agents	6
Office workers	7
Students	6

So there is not any great difference in the frequency of this lesion in the various occupations.

LOCALISATION

In my material the lesion has been about twice as frequent on the right side as on the left. This is rather peculiar in view of the fact, that amongst the cases published in the literature the lesion is about equally frequent on both sides.

In my 51 patients the localisation of the lesion was:

Right side	33	cases.
Left side	17	„
First right side, then left	1	case.

PAST HISTORY OF PATIENTS

The great majority of these patients gives a past history of good health. None of them has ever suffered from relapsing bronchitis or asthma that might give rise to emphysema. Going through the history of each patient with a view to the possibility of previous tuberculous infection, one finds in this material but very few data suggestive of this, considering that practically every adult in a civilised country has gone through a tuberculous infection some time or other.

Pirquet's test has been made only in 22 out of the 51 patients, with the following results:

Positive reaction in 12 (No. 2, 3, 7, 11, 16, 21, 29, 38, 42, 43, 45, 51).

Doubtful reaction in 1 (No. 12).

Negative reaction in 9 (No. 1, 8, 14, 27, 32, 33, 47, 48, 49).

No particular importance is to be attached to the negative reactions, as the test was made only once, as a rule. Yet in No. 8 the test was made 3 times, all with negative result. Also the literature contains a few cases of this form of pneumothorax, in which the patients showed a negative tuberculin reaction (Chauffard, Minkowski, Joerdens, v. Teubern (case No. 3), Enneking (case No. 3), Lassen.)

In the 48 cases that give data on the family history, we find 8 patients who have been exposed to tuberculous infection from near relatives or from other consumptives with whom they have been in close contact (No. 8, 9, 16, 21, 24, 32, 43, 45); of these, No. 8 and No. 32 gave a negative reaction on the Pirquet test. 5 other patients give information of tuberculosis in distant relatives, whom the

patients had seen but very little, or not at all (No. 22, 41, 46, 48, 51).

The previous health of the patients themselves has as a rule been excellent.

42 out of the 51 patients give a past history of good health, that is, leaving out of account that 9 of these patients have previously been ill with lesions that have had nothing to do with pneumothorax. 2 of the patients (No. 2 and 15) have previously been ill with ailments of obscure nature. *None of the 51 patients has ever suffered from pulmonary tuberculosis, but 7 of them give a past history of other tuberculous lesions, namely:*

Scrofulosis in 2 cases: No. 3 had scrofulosis at the age of 16—19 years. Spontaneous pneumothorax 19 years later.

No. 8 had scrofulosis at the age of 9 years. Spontaneous pneumothorax 13 years later.

Pleurisy in 5 cases: No. 4 had pleurisy at the age of 21. Pneumothorax 22 years later.

No. 19. Exudative pleurisy on the left side at the age of 21. 8 years later, pneumothorax on the left side.

No. 32. Chronic pneumothorax on the left side ascertained at the age of 19, but this lesion has probably set in when the patient was only 5—6 years old. At the age of 9 years, the patient is said to have had pleurisy on the right side, lasting 3 weeks.

No. 35. Exudative pleurisy on the left side at the age af 12.

Four years later, first attack of pneumothorax on the right side.

No. 38. Exudative pleurisy on the left side at the age of 11. 11 years later, pneumothorax on the right side.

So, leaving out No. 32, the other 6 of these patients have had a tuberculous lesion a number of years *prior* to the spontaneous pneumothorax.

If then we examine the *X-ray plates* for signs of previous latent diseases of the lungs, it is found that *only 5 out of the 51 patients show distinct cicatricial changes in the lungs* (No. 19, 30, 35, 39, 43). The most pronounced instance of this is found in No. 35, who, at the age of 12, had an attack of pleurisy on the left side, and in which case the left lung shows scattered calcifications (cf. fig. 13, 14, 15). No. 19 showed calcifications in the hilus shadow on the right side and some infiltrations in the right apex. No. 39 and No. 43 showed fibrous scars in the apices; and No. 30 showed several spots in the hilus region and streaks in the lung tissue.

In 14 other patients (No. 8, 9, 10, 14, 15, 17, 20, 21, 22, 27, 28, 36, 48, 49) *the X-ray plates show minor changes* as calcified primary foci, slight enlargement of the hilus shadows, or a slight veiling of the apices, altogether minor changes that are not differing particularly from the usual findings in other persons of clinically perfect health.

In only 6 out of these 51 patients do the X-ray plates show adhesions between the collapsed lung and the parietal pleura (No. 3, 13 (?), 28, 30, 39, 49). This was most pronounced in the case of No. 49 where — as shown in fig. 12 — there is a long, slender adhesion between the apex and the cupola of the pleural cavity, so that the apex is suspended by this strap-like adhesion.

Recapitulating this review of the histories and the X-ray

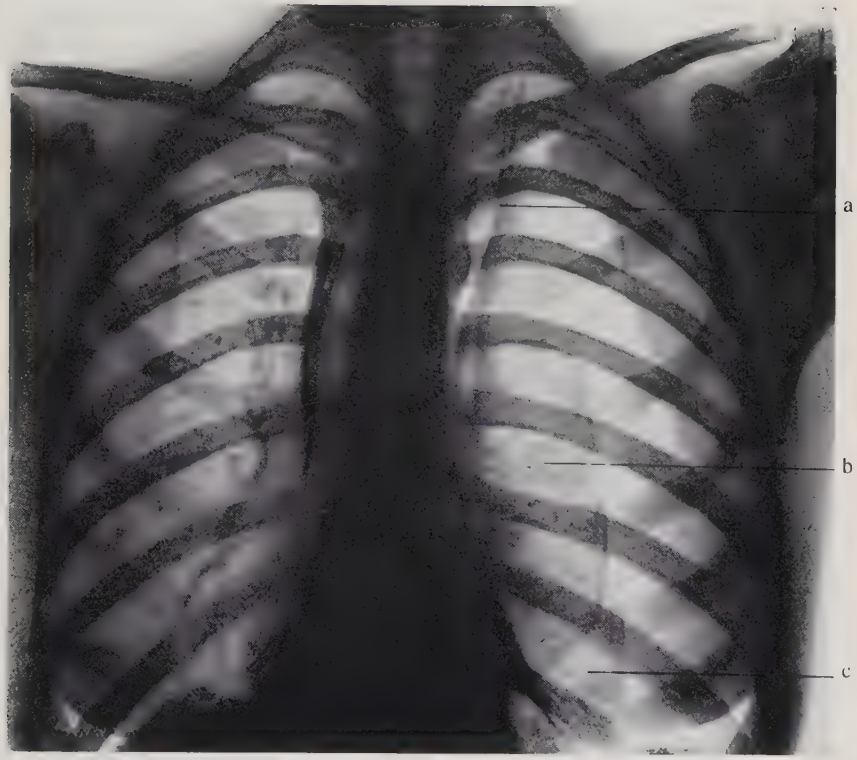

Fig. 12.

Pneumothorax on the left side, with slight displacement and apical adhesion (Pt. No. 49).

a: Adhesion. b: Upper lobe of left lung. c: Lower lobe.

plates in these cases, there is *evidence of cicatricial changes on the surface of the lungs in only 12 out of these 51 patients* — history of pleurisy, calcifications or adhesions on the X-ray plates — namely, No. 3, 4, 13, 19, 28, 30, 32, 35, 38, 39, 43, and 49.

It does not seem far-fetched, then, to imagine that in these 12 patients valve vesicles may have developed next to these scars or adhesions (scar-tissue vesicles).

In the 6 patients who give a past history of scrofulosis or pleurisy, these lesions set in a number of years (4—22 years) prior to the attack of pneumothorax. It is therefore reasonable to assume that the development of the scar-tissue vesicles, and the time that goes before their rupture, take several years.

The remaining 39 cases show no clinical evidence of cicatricial changes on the surface of the lungs. But such changes may very well be so small that they cannot be demonstrated on X-ray examination. In fact, it requires but a very small scar for the production of a scar-tissue vesicle (cf. fig. 5). On the other hand, it may also be that in some of these patients, particularly the ones in the group of negative Pirquet reaction, the spontaneous pneumothorax has been due to the rupture of an emphysematous valve vesicle.

SYMPTOMATOLOGY

ONSET OF LESION

The usual initial symptoms are a sudden *stitch in the chest* and *shortness of breath.*

Although spontaneous pneumothorax in most instances sets in suddenly, its onset may now and then be quite insidious, a fact that has been pointed out especially by Pepper. Still, the latter way of onset is very rare in spontaneous pneumothorax in apparently healthy persons.

In the great majority of cases, the disease sets in quite suddenly, as a rule so well-defined that the patients may often give the hour they took ill.

My material comprises 61 attacks of pneumothorax, and in 54 of these the disease set in suddenly.

In the remaining 7 cases, the onset of the lesion was as follows:

Not particularly sudden in 3 persons who were otherwise perfectly well (No. 15, 22 and 42 *c*), and

in 2 persons who at that time had a cold with coughing (No. 8 *a* and 42 *a*).

In 2 cases, No. 32 and No. 35, the disease was found accidentally, the patients being admitted to the hospital with some other lesion.

The last of these patients, No. 35, gave a history of a previous acute, undiagnosed, attack 6 years before, and probably several slight attacks in between. Here the last attack is therefore to be considered a relapse. It developed so slowly, that it took 1—2 months before the accidentally discovered, coat-formed, pneumothorax had progressed to a pneumothorax with large displacement. This progressive development is illustrated very well in fig. 13, 14 and 15.

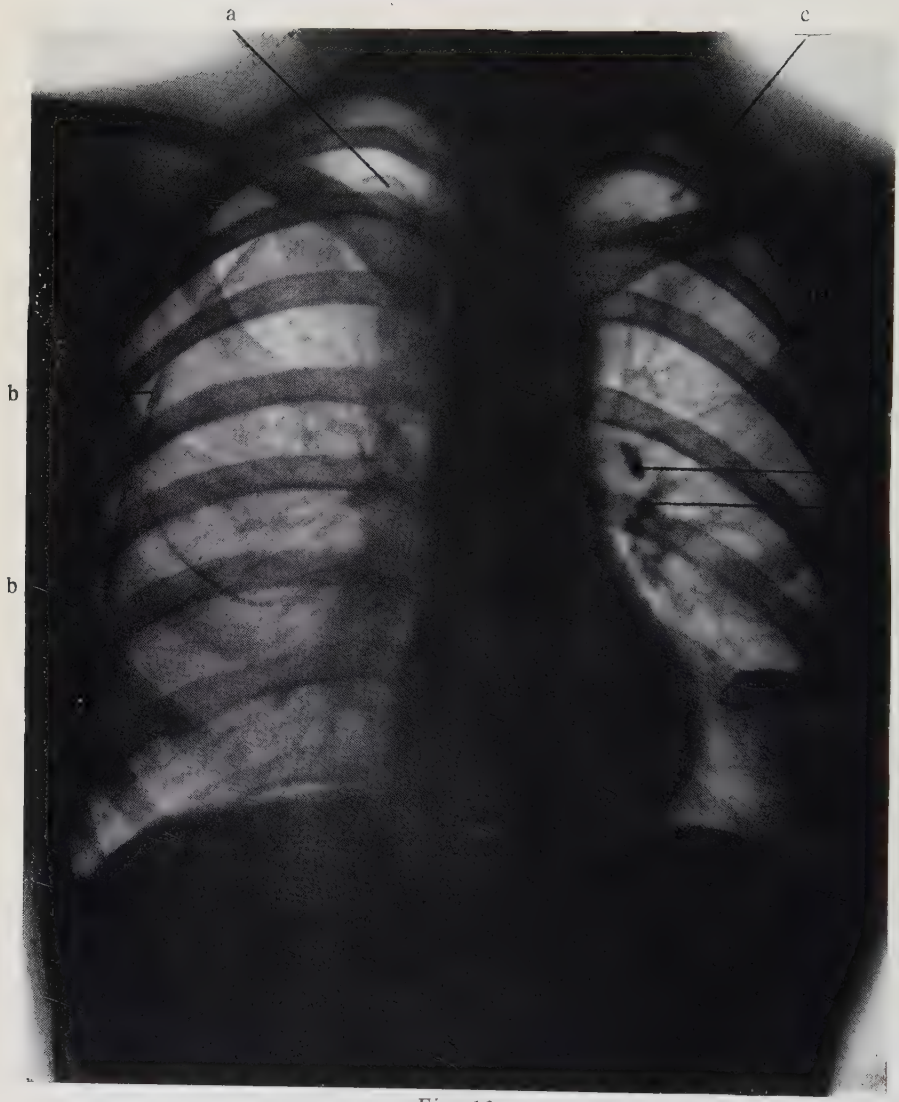

Fig. 13.

Right-sided coat-formed pneumothorax, slowly progressing (Pt. No. 35).

a: Apex of right lung. b: Border of the lung. c: Calcifications in the left lung. (cf. fig. 14 and 15.).

Fig. 14.

Same case as in fig. 13, taken 3 weeks later (Pt. No. 35). Moderate distension of the right side of the chest and flattening of the diaphragm. a: Shadow of right lung. (cf. fig. 13 and 15.).

Between the first two pictures is an interval of 3 weeks. During this period, the lesion has progressed from coat-formed pneumothorax to total pneumothorax with moderate displacement, where the lung is not yet completely compressed. The third plate (fig. 15), which is taken 8 weeks after the pneumothorax was discovered, shows that the lesion has now developed into a severe degree of displacement pneumothorax.

This case has thus offered a unique opportunity to watch how exceedingly slow and insidious the development of an extreme pneumothorax may sometimes be. It is reasonable to assume that the slow development of the lesion in this case is due to a minute size of the perforation — and, probably, this tiny perforation has not been patent continuously. It is the more surprising, therefore, that in this case the lesion went on to chronic pneumothorax (see p. 83.).

Such a slow development of the disease as encountered in this case has not been described before.

In most instances, the disease makes its appearance without any accidental cause whatever.

In the present material, 48 attacks came on without any accidental cause — indeed, 10 of these made their appearance while the patients were lying down.

Only in 13 instances was there any apparent connection between the onset of the disease and some accidental cause. In 11 of these cases, the lesion has made its appearance during physical exertion or shortly after (No. 7, 9, 18, 19, 26 *a*, 26 *b*, 41 *a*, 42 *b*, 42 *c*, 49, 50), whereas in 2 instances the disease developed less acutely in patients who were suffering from a cold, and in whom the coughing spells have possibly been the accidental cause of the pneumothorax (No. 8 *a* and 42 *a*). *This finding, that in most instances there is no accidental cause whatever, is not evident from the literature on this subject.* In most of the cases

reported, the authors have been able to demonstrate an accidental cause, as a rule some form of physical exertion. C. Friesdorf (1927) has tabulated 177 cases from the literature with a view to the accidental cause of the disease, and in these cases some accidental cause was established in about three fifths of the total number.

It is rather peculiar that in 4 cases reported in the literature, the spontaneous pneumothorax has made its appearance during the latter part of pregnancy (Wilks, Bull, Duhot, Szenes). As pointed out by Szenes, pregnancy may probably play a rôle as accidental cause, the respiratory mechanism being altered during the latter part of the gestation.

When my material, in this respect, gives a result that differs from what is obtained by tabulating the cases from the literature, it is undoubtedly because the great majority of the published cases have been instances of very pronounced pneumothorax — some of the cases with tension pneumothorax — and because such severe cases may presumably arise more readily in connection with physical exertion.

As mentioned, in all the acutely developing cases of this disease, the first symptom is a sudden stitch in the chest. This initial pain, which is due to the rupture of the vesicle, should be distinguished from those pains which subsequently, in moderate and severe cases, appear together with dyspnoea and displacement of the mediastinum and the heart. These pains are felt as an increasing tension within the chest, an oppression back of the sternum or in the precordium, or an oppression in the epigastrium.

The initial pains are subject to great variation of intensity. As a rule, they are felt as a cutting stitch, but they vary from a rather slight pricking sensation or a vague oppression in the chest to violent piercing pains. A sudden onset of displacement pneumothorax is always associated with intense pains, but otherwise the intensity of the pains is not proportional to the amount of air in

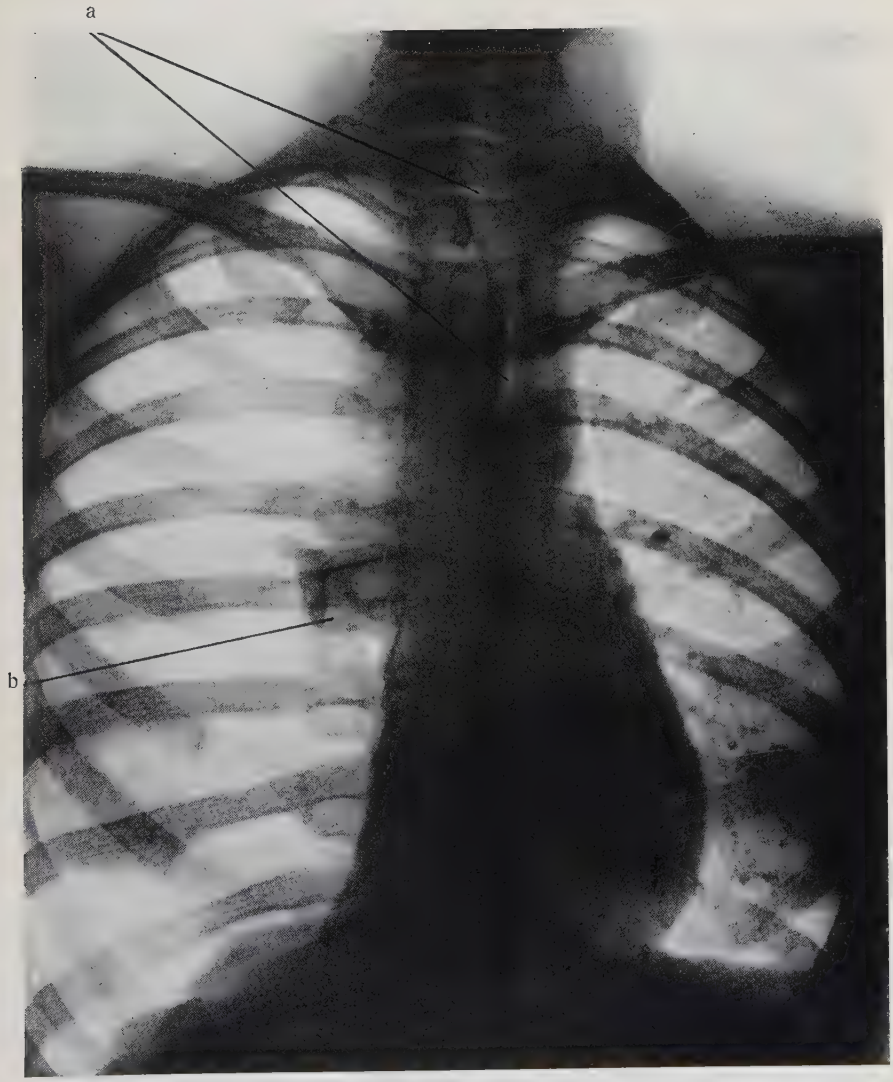

Fig. 15.

Same case as in fig. 13 and 14 (Pt. No. 35), taken 8 weeks after fig. 13. There is now a marked distension of the right side of the chest, with displacement of the organs.

a: Trachea. b: Indistinct shadow in the hilus region.

the pleural cavity, as even a very slight case may be accompanied by a very painful initial stitch.

As other pains arising from lesions of the pleura, the initial pains are aggravated by the respiratory movements, especially by deep respiration and by coughing. In most instances, the pains subside rapidly, disappearing as a rule in a day or two, except in cases of displacement pneumothorax or tension pneumothorax.

The localisation of the initial pains is somewhat varying and indefinite.

Leaving out 4 cases which were not accompanied by pain (No. 8 *a*, 13, 32, 50) and case No. 48, which is in a class by itself, there remain 56 attacks of pneumothorax.

In these 56 cases, the localisation of the initial pain has been as follows:

1. Pains in the same side of the chest ..... 36 cases.
2. Pains in the same side of the chest, radiating out in the back or under the scapula ... 8 „ (No. 2, 12 *a*, 18, 23, 29, 31, 41 *c*, 43).
3. Pains in the chest, not localised to either side 3 „ (No. 5, 10, 40).
4. Pains in the shoulder on the same side ..... 2 „ (No. 12 *b*, 35).
5. Pains in the shoulder and arm on the same side ..... 1 „ (No. 22).
6. Indefinite pains in the back and chest ..... 1 „ (No. 45).
7. Pains in the back, on the same side ..... 2 „ (No. 17, 44).
8. Pains in the back, not localised to either side 2 „ (No. 6, 27 *a*).
9. Pains most marked in the upper part of the abdomen on the same side ..... 1 „ (No. 26 *a*).

56 cases.

As is evident from this survey, in some of the cases the localisation of the pains has been rather confusing. This is the same as is sometimes seen in other lesions of the pleura and in pneumonia, especially in children. These cases may therefore give considerable diagnostic difficulties, which will be discussed in a subsequent chapter.

The other initial symptom is the dyspnoea. This is not by far so frequent as the pains, and often it is absent in the beginning of the disease. When present as an initial symptom the dyspnoea is superficial, a reflex phenomenon, brought about by the pains, and it may be observed even in slight cases when the pains are severe. This is not to be mistaken for the dyspnoea which develops subsequently in the severe cases as a result of the collapse of the lung and the displacement. These points will be discussed in the following under each particular form of the lesion.

Coughing and palpitation of the heart are not amongst the common symptoms, but they may be present. The palpitation may occur in mild cases (No. 46 *a*) as well as in severe (No. 12 *a*).

Amongst the more rare initial symptoms, there may be faintness and pallor when the disease sets in suddenly and is accompanied by severe pains. This is illustrated by No. 41, whose first attack was very painful, whereas the next attack — 2 years later — was not accompanied by any pains worth mentioning.

In a couple of cases (No. 16 and 39) the patients have brought up, in the beginning of the disease, a little blood-tinted mucus, probably resulting from an insignificant hæmorrhage at the rupture. But, a large hæmoptysis, such as observed by Szenes, is undoubtedly an extremely rare phenomenon.

Vomiting has been present in the beginning of the disease only in 2 of the cases (No. 2 and 40); and only in 1 case (No. 24) was the lesion ushered in by hiccough.

CLINICAL FEATURES

What groups these cases into one nosographic entity is the fact, that they are cases of pneumothorax where the clinical examination fails to reveal any etiological factor.

The clinical appearance of these cases, on the other hand, is subject to wide variations of severity, all degrees of spontaneous pneumothorax being represented, ranging from very slight, abortive, cases of partial pneumothorax to the most severe instances of critical acute tension pneumothorax. Exceptions from the rule in this material are 4 cases of chronic pneumothorax, 1 case of hæmopneumothorax and 1 case of spontaneous pneumothorax with marked subcutaneous emphysema. These important variations of the clinical picture will be mentioned separately. In all the other 45 patients, in whom 55 attacks of pneumothorax have been observed (8 patients having more than one attack) we meet with a series of more or less severe attacks of pneumothorax, wherein all transitions are represented.

The one factor which dominates the clinical picture of the lesion, is the amount of air that is forced out into the pleural cavity from the ruptured lung, for it is the differences in the amount of the pneumothorax air and the pressure it produces in the pleural cavity that give rise to all the differences in the clinical features of the individual cases. It should be mentioned, however, that the clinical picture of the lesion is largely dependent upon the rapidity with which the pneumothorax is established in the respective cases, whether it is very acute in its appearance or it develops more gradually. For the more rapidly the pneumothorax is established, the more violent are the symptoms.

Even though there are very gradual transitions from the mildest cases to the most severe, it is yet of some practical value to divide these attacks of pneumothorax after certain types, as each of these groups presents a rather considerable uniformity of clinical features.

From the amount of air in the pneumothorax cavity, one may then distinguish between:

1. Partial pneumothorax (6 cases in my material).
2. Coat-formed pneumothorax (3 cases).
3. Total pneumothorax without — or with very slight — displacement (22 cases).
4. Total pneumothorax with moderate displacement (14 cases).
5. Tension pneumothorax (10 cases).

This classification represents the various degrees of the lesion, and it gives just as well an expression of the various stages of the disease.

Some of the cases pass through one stage after another, so that the clinical picture is gradually progressing from a mild instance to a severe case. The stage at which an attack of spontaneous pneumothorax is checked, of course, is largely dependent upon the character of the perforation; but it is also of considerable consequence whether the patient goes to bed immediately after the attack, or whether he keeps on going about and works after the attack, for in the latter case the lesion is often aggravated considerably.

It is but seldom one meets the classical picture of spontaneous pneumothorax with collapse, severe pains and considerable dyspnoea, associated with marked distension of the respective side and marked displacement of the heart and diaphragm — this is seen only in the relatively few cases of true tension pneumothorax, which was found in 10 cases of this material.

The characteristic of the great majority of these pneumothorax cases is the very fact they are moderate and benign attacks of pneumothorax. This fact is rather peculiar in view of the lesion being due to the rupture of a valve vesicle, and that, consequently, this form of pneumothorax is always a valve pneumothorax.

A very essential feature in the clinical picture of the spontaneous pneumothorax in the apparently healthy is the fact that the lesion is not associated with any rise of the temperature. Still, this does not apply quite absolutely to all cases. As a rule, the patients have on the first day of illness either a perfectly normal temperature or, what is seen most frequently, a very slight rise of the temperature in the evening (37.7 — 38.1). During the first week of illness, too, the temperature is often elevated a few tenths of one degree, but after this it always stays perfectly normal throughout the further course of the lesion.

However, patients with hæmopneumothorax form an exception to this rule. Also tension pneumothorax is often associated with considerable rise of the temperature, but this subsides in a few days.

Thus, when No. 12 *a* was admitted to the hospital, 4 days after the onset of his illness, he had a temperature of 38.9, but this fell the next day to 37,2, and it remained normal throughout the remainder of his stay in the hospital.

No. 27 *b,* who was admitted 12 hours after the onset of the illness, showed a rise of the temperature the first 3 days — maximum 38.8.

No. 34 had a rise of the temperature lasting 6 days — maximum 38.8.

No. 11 had a rise of the temperature through 5 days — maximum 38.6 — and one evening later, on the 10th day after the onset, when the temperature went up to 37.8.

These 4 cases were all cases of tension pneumothorax without the slightest exudate in the pleural cavity. In each of them, the temperature stayed perfectly normal throughout the further course of the disease.

One case of partial pneumothorax with very slight displacement (No. 39) had a rise of the temperature during the first week of illness; afterwards, the temperature was perfectly normal. In this case, the X-ray examination

revealed a very small amount of fluid in the phrenocostal groove.

One case of total pneumothorax without displacement (No. 47) showed a rise of the temperature the first 6 days — maximum 38.3 — then normal temperature. No exudate could be made out in this case (see fig. 18 and 19).

In all the other patients, the temperature has been normal throughout the period of illness, apart from a slight initial rise on the day of admission (37.7 — 38.1), or a very slight elevation (37.7 — 37.9) on the following days.

Another important feature in the symptomatology of these pneumothorax cases is the fact that they proceed without any sign of pleural irritation. Apart from cases with bleeding into the pleural cavity, these patients show no pleural exudate that may be demonstrated on auscultation, nor do the X-ray plates as a rule show the slightest sign of pleural effusion.

In this material, we find 10 cases wherein there was a small exudate which could not be demonstrated clinically and could be made out only on the X-ray plates (No. 5, 6, 13, 20, 25, 33, 35, 38 *a*, 39, 41 *b*). In 8 of these cases the X-ray plates showed a shadow of merely a minimal amount of fluid in the phrenocostal groove (cf. fig. 23).

In one case, No. 36, the X-ray plates (fig. 21 and 22) showed the presence of a fluid which on puncture proved to be blood.

All the other cases showed no sign whatever of even the smallest amount of fluid, neither at the onset of the illness nor subsequently.

Friction sounds constitute another sign of pleural irritation, and they are practically always absent in these cases.

Only in 2 cases could friction sounds be made out in the beginning of the disease (No. 1 and 21). These patients are discussed on p. 46.

In 2 cases, friction sounds were heard after the lung had expanded (No. 5 and 12 *b*).

In 1 case, friction sounds were heard 7 weeks after the onset of the illness (No. 25).

And in 1 case of chronic pneumothorax, friction sounds were noticed 9 months after the discovery of the lesion (No. 35).

In all the other cases, no friction sounds could be made out at any time during the illness.

A couple of the patients (No. 24 and 41 *b*) have complained of pricking discomfort in the chest during the period the lung was expanding; but there were no friction sounds in these cases.

Returning to the types of pneumothorax set up above, in the following I shall try to describe the characteristic features of each type seperately.

PARTIAL PNEUMOTHORAX

In the great majority of cases, spontaneous pneumothorax in apparently healthy persons makes its appearance in the form of a total pneumothorax, the air extending over the entire pleural cavity. Only a small minority of the cases shows a partial pneumothorax, as only a part of the pleural cavity is filled with air while the lung remains expanded in the other part of the cavity. In some of these cases the partialness of the pneumothorax is due to the presence of adhesions which prevent the air from extending further and keep the lung from collapsing. But in other cases of partial pneumothorax, the partialness is owing to the fact that only a very small amount of air escapes after the rupture of the lung so that only the adjacent section of the lung collapses.

Hitherto, the condition of partial pneumothorax has been known but very little, the literature giving — as far as I have been able to find out — only one instance

of this condition. In 1926, Koelensmid described this case: The patient was an otherwise healthy carpenter who, while drawing a bottle, had a sudden attack of pains in the right side. The Roentgenogram showed a small, walled-off, partial pneumothorax in the right phrenocostal angle, in which adhesions could be made out distinctly. This air space diminished considerably during the next 4 days. Koelensmid thinks that the presence of the adhesions in this case is the reason why the pneumothorax was only partial.

My material comprises 6 cases of partial spontaneous pneumothorax (No. 8 *a,* 21, 39, 45, 46 *a,* 46 *b*), amongst which there is one case, No. 39, wherein the presence of adhesions did undoubtedly prevent the development of a total pneumothorax. In this case the X-ray examination showed that the lung was compressed above and below while its middle third was adherent to the chest wall, filling the entire middle section of the pleural cavity.

In addition, there is one case (No. 21) where a rather large pneumothorax occupied the lower half of the right pleural cavity. 5 months later, this patient had a severe degree of bronchopneumonia which gave rise to pyopneumothorax on the right side, with complete collapse of the lung.

But apart from these cases, *there are unquestionably some cases of partial pneumothorax, where the partialness of the lesion is due entirely to the fact that the rupture of the lung closes so rapidly that only a very small amount of air escapes into the pleural cavity.*

The case of No. 8 illustrates very well that it is not always the presence of adhesions which causes that the pneumothorax is only partial. For this patient had first a very slight partial pneumothorax on the right side, and later she had a large total pneumothorax on the same side, proving that there were no adhesions to keep the lung from collapsing.

These attacks of partial pneumothorax are so mild that,

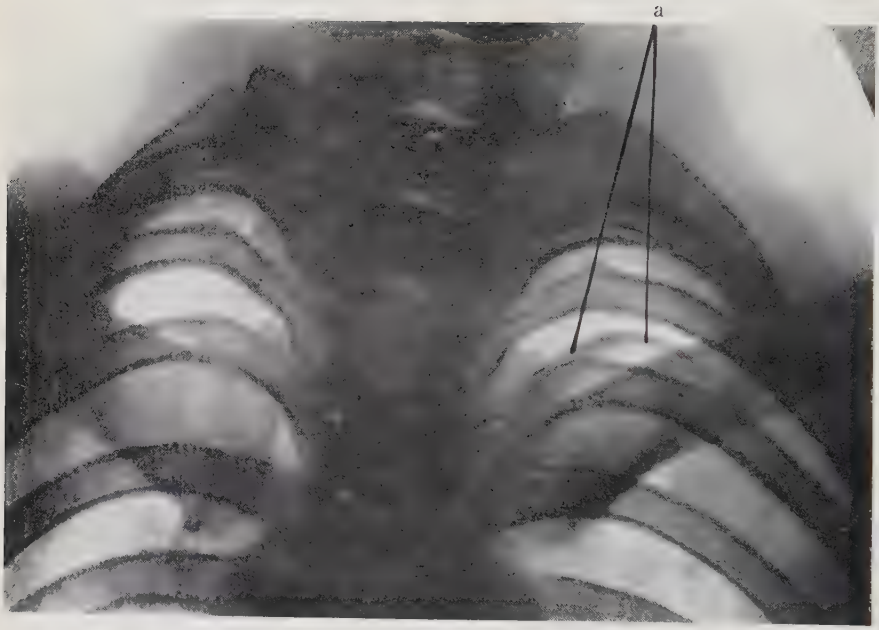

Fig. 16.
Partial pneumothorax on the left side (Pt. No. 46 *a*).
a: Border of left apex.

often, the patient does not consult a physician at all; and the objective findings are so vague and so hard to interpret that the diagnosis would in most cases be impossible to make without X-ray examination.

The lesion has such a marked tendency to rapid spontaneous recovery that all the signs subside completely in 1—2 weeks.

4 of my cases illustrate very well this peculiar, altogether very mild, disease.

No. 46 has had 2 well-observed attacks of very mild spontaneous, partial pneumothorax on the left side, 7 months apart.

The first time, the disease set in with sudden sticking pains around the left curvature. The pains were not particularly severe to begin with, but they were increasing rapidly in intensity. The second time, the stitch was also quite sudden in its onset, and it was more severe. Both times, the stitch was accompanied by dyspnoea, showing as shallow painful respiration. And both times, the patient was free of symptoms the next day when lying quiet.

Physical examination, at the first attack, showed impairment of the respiration on the left anterior surface of the chest, with a faint metallic tinkling note. In the second attack there was only a slight tympany and suppression of the breath-sounds over the left apex to costa III; and these signs disappeared completely in 4 days (in bed).

The X-ray plates, taken 7 days after the onset of the first attack show no distinct sign of pneumothorax in the total picture of the lungs. But, in the special picture of the apices (fig. 16) one will notice that the upper part of the left pleural cupola is filled with air, while the apex of the lung is lowered a few centimeters, presenting itself very distinctly with its arching contour.

In both attacks the temperature was normal throughout the illness, and the patient recovered completely in 2 weeks.

No. 8 is a female teacher. One week before her admis-

sion to the hospital she has been suffering from an ordinary cold, with coryza and cough, but no pain in the chest and no dyspnoea. After she is admitted, the physical examination shows — beside scattered rhonchi over both sides of the back — a resonant percussion tone over the entire right front of the chest, with slight metallic note, and feeble, distant, breath-sounds, together with a resonant percussion tone over the right side of the back. There was, in addition, a subcutaneous emphysema on the right side in front, occupying the supraclavicular fossa and extending down to costa II.

The subcutaneous emphysema disappeared as early as 4 days after; and about 2 weeks after the onset of the lesion, signs of pneumothorax could no longer be made out on X-ray examination.

$8^3/_4$ years later, this patient had total spontaneous pneumothorax on the same side.

The case is peculiar by the absence of pain at the initial rupture and by the presence of subcutaneous emphysema, which will be discussed in a subsequent section.

No. 45 is likewise an example of partial spontaneous pneumothorax, although in this case the air content of the pleural cavity was a little greater than in the preceding cases. It is a young man who has a sudden attack of very severe pains in the chest and back, not accompanied by dyspnoea. 2 days later he applies to the Medical Policlinic of the Rigshospital, where physical examination reveals no abnormalities except that the breath-sounds over the back are less distinct on the left side than on the right.

The X-ray plate (fig. 17), taken 3 days after the onset of the lesion, shows a partial pneumothorax occupying the upper lateral part of the left pleural cavity.

3 weeks later, when the patient returns to the policlinic, he is perfectly well, and neither auscultation nor X-ray examination show any abnormality whatever.

In addition to these well-observed cases of partial pneumothorax, there are 3 other patients (No. 24, 35, 44),

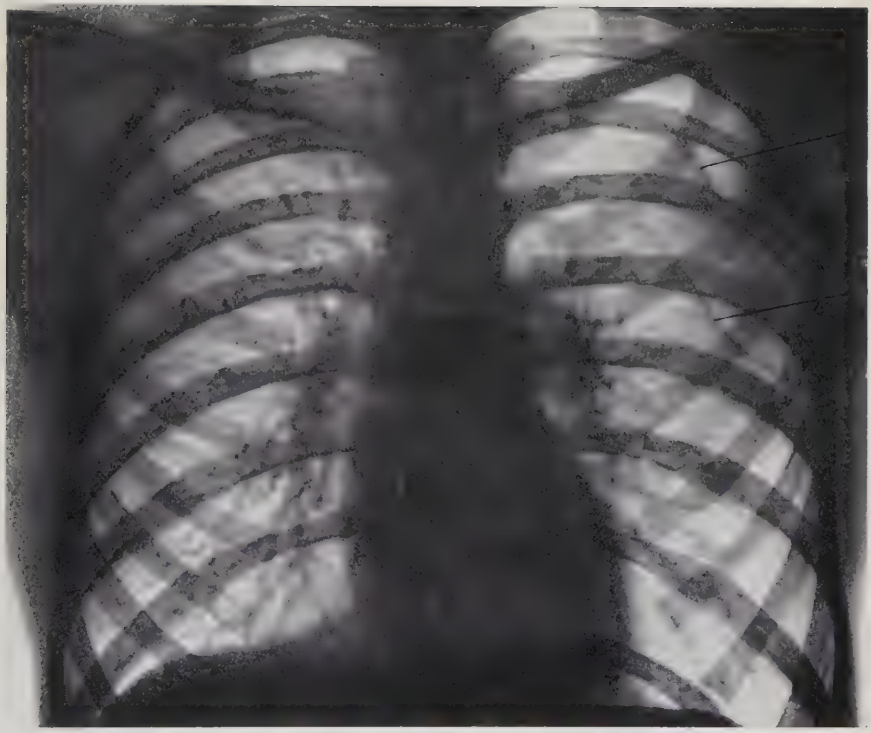

Fig. 17.
Partial pneumothorax on the left side (Pt. No. 45).
a: Border of upper left lobe. b: Border of lower lobe.

with subsequent attacks of total pneumothorax, who give a *past history* of very slight and brief ailments which are undoubtedly to be interpreted as abortive attacks of pneumothorax that have not been recognised as such. 2 other patients (No. 15 and 40) have perhaps had similar slight and transitory attacks.

The characteristics of the partial spontaneous pneumothorax may then be given as follows:

The disease begins with an initial rupture pain and shallow painful respiration.

Auscultation, immediately after the onset, reveals a slight suppression of the breath-sounds and sometimes a slight tympany — usually over one apex, but occasionally (No. 21) over some other part of the chest.

X-ray examination, immediately after the onset, shows partial pneumothorax.

The temperature is perfectly normal; and there are no signs of pleural irritation.

The pains and the respiratory discomfort subside in a day or so; and the auscultation and X-ray examination show that the pneumothorax air disappears in 1—2 weeks.

Some of these patients have sooner or later a relapse — either as partial pneumothorax or as total pneumothorax.

COAT-FORMED PNEUMOTHORAX

The difference between the partial pneumothorax and the coat-formed is merely a difference in degree.

In this condition the X-ray plate shows a moderate amount of air along the peripheral border of the pleural cavity, so that this layer of air envelops the lung like a coat. Throughout the pleural cavity, the lung is retracted from the chest wall, but its surface is only 2—3 cm. from the chest wall. There is no displacement of the mediastinum or the heart (cf. fig. 13).

My material comprises 3 cases of coat-formed pneumo-

thorax (No. 37, 38 *a*, 38 *b*). In No. 37 the lung is retracted more than stated above, the surface of the lung being 4—5 cm. from the chest wall.

In these cases the onset of the illness has been marked by a sudden stitch, but there has been no dyspnoea. The auscultation shows tympany and suppression of the respiration over the whole side of the chest, in front as well as on the back. In one of these cases (No. 38 *a*) the X-ray plate shows a minimal amount of fluid in the phrenocostal groove (angular shadow). In all three instances, the temperature has been perfectly normal throughout.

The stitch has subsided in a few days, and there has been shortness of breath only on rather active movements (walking).

In No. 37 all the signs of pneumothorax disappeared in 3 weeks.

In the first attack of No. 38 it took 2 months before the air had disappeared completely; in the second attack, on the other hand, all the air was absorbed in 2 weeks.

Finally, No. 35 showed at first a coat-formed pneumothorax, but, as mentioned before, this was gradually developing into an extreme degree of displacement pneumothorax (cf. p. 56.).

TOTAL PNEUMOTHORAX WITHOUT - OR WITH VERY SLIGHT - DISPLACEMENT

In these cases the X-ray picture shows a considerable compression of the lung.

In a few instances the lung is pushed flat up against the mediastinum — as illustrated in fig. 18 — but in most cases it is compressed around the hilus, in outline and size about like a fist. The pressure difference between the affected pleural cavity and the healthy is not sufficient to bring about a distinct displacement of the mediastinum or the heart over toward the healthy side.

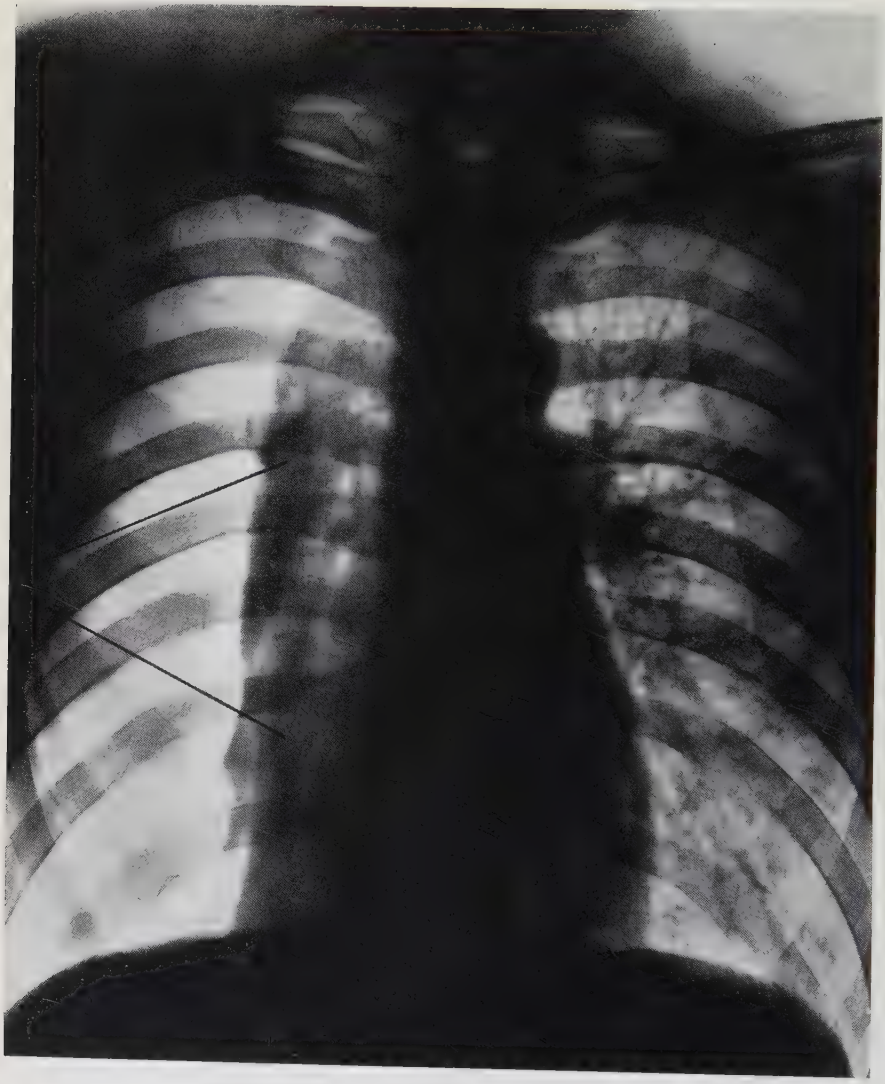

Fig. 18.
Right-sided pneumothorax without displacement. Pt. No. 47.
a: Collapsed lung.

Fig. 19.

Same case as in fig. 18, taken 16 days after. Pt. No. 47.
a: Right lung, beginning expansion.

My material comprises 22 cases of this form of pneumothorax (No. 2, 3, 7, 8 *b*, 10, 15, 18, 22, 24, 27 *a*, 28, 33, 40, 41 *a*, 41 *b*, 41 *c*, 42 *c*, 43, 44, 47, 49, and — probably — No. 17).

In some of these cases the history shows that the symptoms have been developing gradually and, especially that there has been a distinct interval between the rupture pain and the subsequent dyspnoea. It is very likely that in these cases the patients have had at first only a partial or a coat-formed pneumothorax, and that by their walking about this condition has gone on to total pneumothorax with compression of the lung. This applies to cases as No. 27 *a*, for instance.

In this case, No. 27 *a*, the patient had, 2 days before his admission to the hospital, an attack of pains in the back, in the region of the lower dorsal vertebræ, radiating out to the sides, equally to right and left. The next morning he was feeling better, but the pains were aggravated as he was working, so that he had to quit work. On the day of admission the pains have again subsided considerably, but now he readily gets short of breath. Physical examination shows total pneumothorax on the right side with very slight displacement (cf. fig. 27).

Similar features are met with in No. 44. One day this patient had a sudden stitch in the right side of the chest. 2 days later she was short of breath when she woke up in the morning, and during the following week she had difficulty in breathing and stitch in the back when walking on the street or upstairs. On admission to the hospital she had total pneumothorax on the right side with slight displacement.

No. 43 had a sudden stitch in the chest one morning; yet he managed to keep working all day long, in spite of some shortness of breath. The next day, however, he had to quit work, as the dyspnoea was aggravated.

No. 28 woke up in the morning with a moderate stitch in the left side of the chest. During the day the pains were

getting worse, especially on motion, and the patient was becoming rather short of breath. When admitted to the hospital the next day, he had total pneumothorax with doubtful displacement.

In most of the cases, however, the clinical features of the disease have been manifest more rapidly, probably because the perforation has been so wide that a total pneumothorax is established in a very short time.

In some of the patients the initial rupture pains have been accompanied by shortness of breath. This applies to No. 25, 40 and 47.

But, in some cases the *only* subjective symptoms have been the sudden pains in the chest. These patients have not been short of breath at all, and their only complaint is severe pain in the chest. Examples of this are found in No. 2, 10, 18 and 33.

On physical examination it is noticed that the respiratory movements of the chest are smaller on the affected side than on the healthy side. As a rule, however, there is no noticeable distension of the affected side — only a couple of the cases have shown a very slight dilatation.

On auscultation, the percussion note over the affected side is either normal or tympanitic. In some cases the resonant percussion note extends as far down as costa VIII in front and costa XII on the back. The breath-sounds are markedly suppressed. In some cases the feeble breath-sounds have an amphoric quality; in a few cases the breath-sounds have a metallic quality, and on coughing there may be metallic tinkling. The vocal fremitus is greatly diminished. No distinct displacement of the heart can be made out on auscultation.

In none of the cases was the intrapleural pressure measured in the beginning of the disease, but undoubtedly there is only a slight increase.

In case No. 8 *b* the intrathoracic pressure was measured 17 days after the onset of the illness. At this time, the affected lung was compressed to half its normal size, and

there was no displacement of the heart. The manometer showed: ÷ 2½ cm. H_2O and +1½ cm. H_2O.

As a rule the pulse rate is somewhat increased during the first days of illness, but not over 100, and it usually returns to normal in a couple of days. In most of the cases the respiratory frequency is also increased the first days — up to 20—24, or 28 at the most — and this, too, is normal again within a couple of days. The behaviour of the temperature has been mentioned already, on p. 63.

In all these cases, the rule is that the symptoms —the pains as well as the dyspnoea — subside in a couple of days when the patients keep their bed. And even in patients who are walking about, the illness reaches its climax in a few days, whereafter the pains subside, leaving but a slight degree of dyspnoea on movements. An instance of this is found in No. 49.

In some cases, where the patients are staying in bed, one may notice even as early as about 2 weeks after the onset of the illness that the lung is plainly beginning to expand — as shown in fig. 19.

The quickest recovery is about one month, but in most cases it takes from 6 weeks to 2½ months before all the pneumothorax air is absorbed. The last remnant of this air is usually found in the phrenocostal groove or over the apex.

TOTAL PNEUMOTHORAX WITH MODERATE DISPLACEMENT

In cases of this type the X-ray examination shows that the lung is very much compressed, appearing as a lump — the size of a child's fist — on the side of the heart shadow. The affected side as whole is somewhat distended, and there is some dilatation of the intercostal spaces.

The mediastinum and the trachea are somewhat displaced toward the healthy side of the chest, and the level

of the diaphragm is lowered. On the X-ray plate, the lung tissue of the lower part of the healthy lung appears to be more dense than normal — from compression through the displacement of the heart.

My material comprises 14 cases of this type (No. 1, 5, 9, 13, 14, 19, 20, 25, 29, 31, 42 *a*, 42 *b*, 50, 51).

Patients with displacement pneumothorax will always feel somewhat short of breath.

In a few of these cases the anamnesis shows that the patients have not had displacement pneumothorax from the very onset of the illness.

No. 29 is an example of this. To begin with, the patient felt only some stinging pains, but she was not short of breath the first 5 days, while she was staying in bed. Then she got up and went to her doctor, and now the symptoms of a marked displacement pneumothorax appeared (fig. 20).

In two of the cases, No. 14 and 42 *a*, where the patients have applied to the hospital rather late, ca. 2 months after the onset of the illness, there was no dyspnoea in the beginning of the disease, and it came on gradually. In these cases the displacement has probably been developing gradually, too, as even a slight pressure difference between the two pleural cavities, persisting for a length of time, will effectuate a considerable displacement.

In all the other 12 cases the disease begins at once with pains and dyspnoea. In these cases, however, the dyspnoea is merely a moderate shortness of breath. The physical examination shows diminished respiratory movements and some distension of the affected side.

In addition, there is a demonstrable moderate displacement of the heart. In the right-sided pneumothorax the ictus is pushed out in the papillary line or a little further out. In left-sided pneumothorax the absolute heart dullness has disappeared and the heart sounds are sometimes heard most distinctly at the right of the sternum.

There is tympany of the affected side. Here the breath-

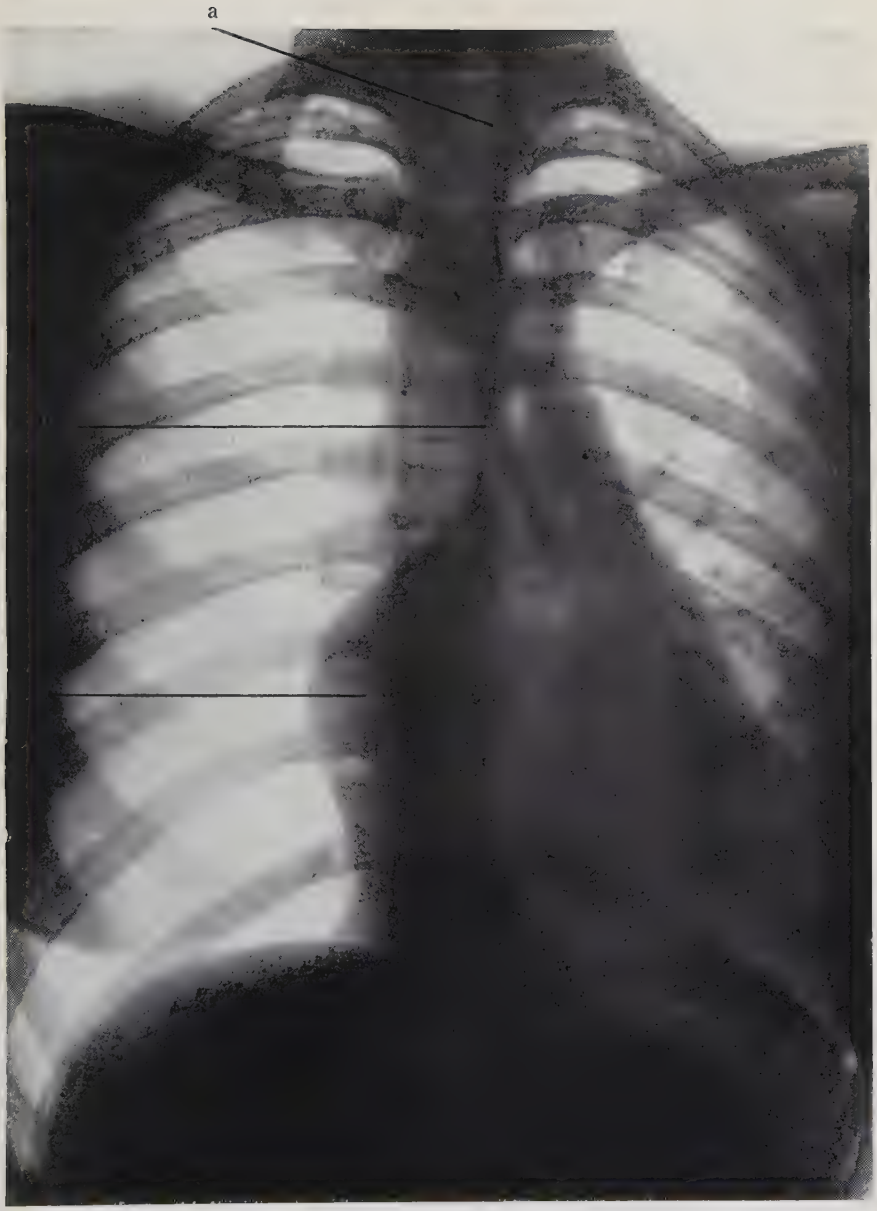

Fig. 20.
Right-sided pneumothorax with rather marked displacement.
Pt. No. 29.
a: Displacement of the trachea. b: Displacement of the mediastinum. c: Right lung.

sounds are markedly suppressed, sometimes entirely absent towards the base. In some cases the respiration has an amphoric quality, and occasionally a metallic tinkling.

The intrapleural pressure is measured in 5 of these patients:

No. 42 b: Pressure measured 7 days after onset of illness: + 4 cm. H_2O + 10 cm. H_2O.

No. 31: Pressure measured 4 days after onset of illness. Marked compression of the lung. Moderate displacement of the heart. Average: + 1 cm. H_2O.

No. 20: Pressure measured 13 days after onset of illness. ÷ 2 cm. H_2O + 2 cm. H_2O. Marked compression of the lung. Moderate displacement.

No. 25: Pressure measured 16 days after onset of illness: Average: ÷ 3½ cm. H_2O. Complete compression of lung. Moderate displacement.

No. 51: Pressure measured 35 days after onset of illness: ÷ 8 cm. H_2O ÷ 4 cm. H_2O.

In the first days of the illness there is usually an increase in the frequency of the pulse and respiration, which soon become normal again. In No. 42 *b* and in No. 51, however, the pulse was perfectly normal. 3 days after the onset of the illness, No. 31 showed: Pulse 84, and Resp. 24.

In all the other patients *admitted some time after* the onset of the illness, the pulse rate is either normal or accelerated a very little, 88 at the most; and the frequency of the respiration is 16—20, 24 at the most.

The course of the disease is about the same as in total pneumothorax without displacement, the initial pains and the initial dyspnoea being checked in a couple of days if the patient goes to bed at once. But even in the patients who continue to walk about, the symptoms subside gradually, leaving merely a moderate dyspnoea on movements.

Under proper treatment it takes from 6 weeks to 2—3 months before the pneumothorax air is completely absorbed. 4 of the patients (No. 5, 14, 19 and 42 *a*) did not come under treatment till 1—2½ months after the onset of the lesion, but, nevertheless, in these cases the air was absorbed just as rapidly as in the other cases.

TENSION PNEUMOTHORAX

It is only in a relatively small number of cases of spontaneous pneumothorax in apparently healthy persons that the lesion goes on to become a true tension pneumothorax.

This condition is characterised by the exhausted condition of the patient, in whom a marked displacement of the mediastinum and the heart and the resulting compression of the healthy lung bring about a very marked air-hunger. This is associated with symptoms of collapse, tachycardia, pallor and sweat, and finally — as a sign of functional failure of the respiration — a more or less pronounced degree of cyanosis. This is the classical picture of the disease with collapse, pains and dyspnoea we are here meeting with.

The steady increase in the intrapleural pressure on the affected side may be due to various factors. Sometimes it is due to the fact that the patients keep on working after the onset of the illness; sometimes it is due to coughing; and, finally, there are some cases with severe pains where the patients are breathing with groaning expiration through the glottis partly closed from reflex action, so that the result is a more or less continuous succession of abrupt pressure increases in the respiratory passages, forcing the air out through the ruptured valve vesicle.

The most severe degrees of acute pneumothorax as sometimes encountered in patients with bilateral pulmonary tuberculosis do not occur in spontaneous pneumothorax in apparently healthy persons, as the prognosis of the lesion itself is far better in the latter group of patients,

partly because the other lung is healthy, and partly because these patients are nearly always young persons without any heart lesion.

My material comprises 10 cases of tension pneumothorax (No. 11, 12 *a*, 12 *b*, 16, 26 *b*, 27 *b*, 30, 34, 36 and, most probably, 26 *a*).

The physical examination of these patients shows all the signs of pneumothorax fully developed together with an enormous distension of the affected side and a marked displacement of the heart.

In extraordinary cases, as in No. 26 *a*, the initial symptoms of right-sided tension pneumothorax may be so deceptive that the condition is mistaken for an acute abdominal lesion (perforated gastric ulcer).

As mentioned before, the initial stage of tension pneumothorax is often accompanied by a rise of the temperature, but this subsides again in a few days (cf. p. 63.).

In most cases, the lesion sets in acutely with severe pains, followed shortly after by marked dyspnoea; and the lesion progresses so rapidly that the tension pneumothorax is established as early as in the first day of illness.

Occasionally, however, one meets with patients who are walking about with a spontaneous pneumothorax that does not trouble them very much to begin with, but *gradually, within a few days, it goes on to tension pneumothorax.*

This has been the case in 3 of my patients.

No. 36 woke up in the night, 2 days before his admission to the hospital, with very severe pains in the left side of the chest. The pains subsided very quickly, and he went to sleep once more.

Since then the patient was suffering from a steadily increasing dyspnoea. Yet he was able to keep working, until he was admitted with pronounced symptoms of tension pneumothorax, *marked dyspnoea and slight cyanosis* (temperature 38.7).

The X-ray plate of this patient (fig. 21), taken on the day of admission, shows a very pronounced tension pneu-

mothorax on the left side with a distinct surface of fluid on the bottom of the pleural cavity. Puncture was made at once, which relieved the patient considerably. By the puncture escaped a large amount of air, with a few squirts of blood.

The next X-ray plate (fig. 22), taken 5 days later, illustrates very plainly that after the puncture the heart has returned almost to its normal position.

No. 12 *b* is the case of a patient who took ill 4 days before he was admitted to the hospital with pains under the right scapula. Still he was able to keep working till the day before his admission to the hospital, when the pains increased, and he was getting short of breath; in addition, he now had pains in the precordium, and he was feeling very poor. He was coughing a good deal, but brought up only a little expectorate without blood.

On admission, the patient was *rather pale, slightly perspiring and markedly dyspnoeic.* Temperature 38.9; pulse 108; respiration 44. There was displacement of the heart as well as of the liver.

No puncture was made in this case, and even the next day the patient was less dyspnoeic when lying quietly; and now the temperature was 37.2, pulse 88 and respiration 26.

No. 30 took ill 4 days before he was admitted to the hospital, with a stitch in the right side of chest and marked shortness of breath. The dyspnoea was increasing the first 2 days, and it kept constant the next 2 days, so that it prevented any strenuous physical work. Even when lying quietly he is troubled considerably by dyspnoea, and the stitch in the right side is constantly present. He has had some dry coughing. On admission to the hospital, the patient is *slightly cyanotic.* Temperature 38.1; pulse, regular and full, 104—108; respiration 32—34. Puncture is not performed, and the patient is better even the next day.

In the other 6 cases the records show that the tension pneumothorax has been fully established even within the first 24 hours.

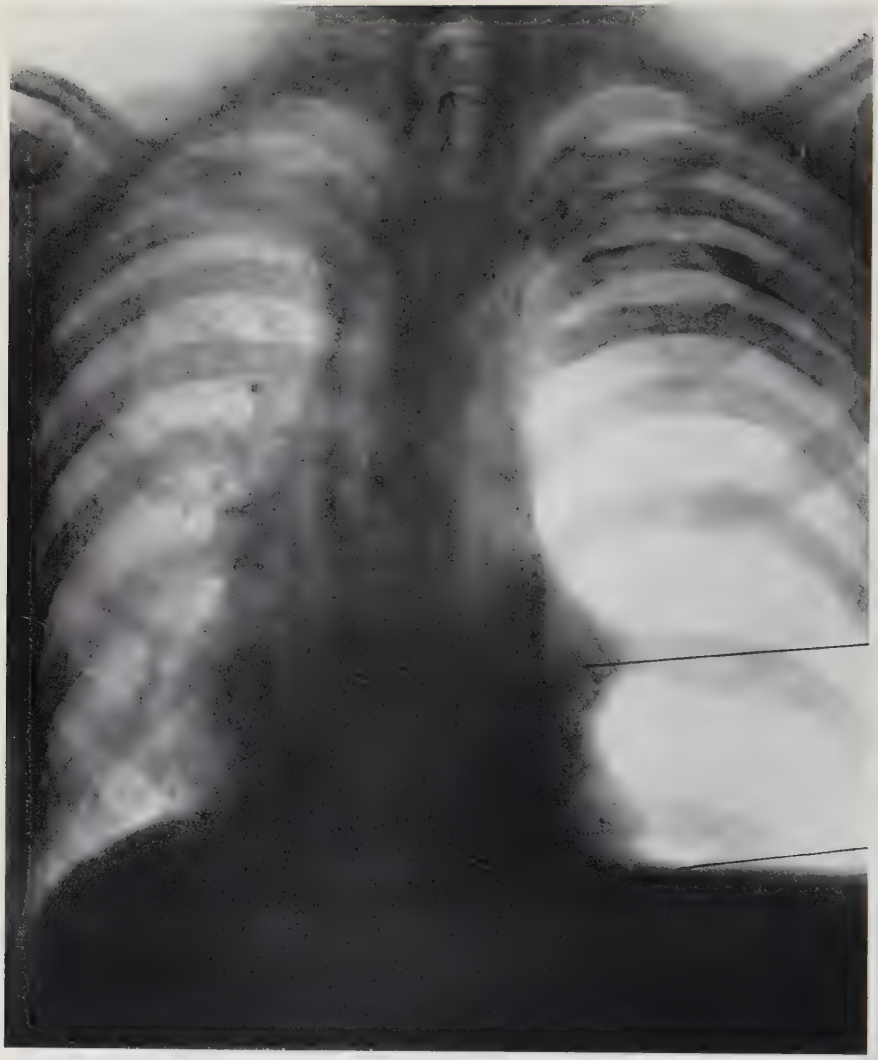

Fig. 21.

Tension pneumothorax on the left side, with a small hæmorrhage. Pt. No. 36. Note the marked distension of the intercostal spaces and the displacement of the heart over on the right side!
a: Left lung. b: Surface of the intrapleural hæmorrhage.

Fig. 22.

Same case as in fig. 21, taken 5 days after the puncture. Pt. No. 36.
Note how the heart has moved back again!
a: Left lung. b: Surface of the intrapleural hæmorrhage.

No. 12 *a* was taken ill at 3 o'clock in the morning, with pains in the chest. Later in the day he had dyspnoea, precordial pains, marked palpitation of the heart, and he brought up a little tenacious phlegm. On admission, in the afternoon, he is *pale, slightly perspiring and somewhat dyspnoeic.* Temperature 37.6; pulse 84, regular; respiration 36. Puncture is not performed, and the next day the patient is considerably improved: Temperature 37.6. Pulse 72, regular. Respiration 20.

No. 16 takes ill suddenly in the evening, with violent pains in the chest; he coughs and brings up a little expectorate with sanguinolent streaks. In addition, he is *very short of breath, shivering and sweating profusely, very weak and uneasy.* Temperature 37.3. When admitted to the hospital, the next day, he shows *very marked dyspnoea and slight cyanosis.* Temperature 37.5. Pulse 96, strong. Respiration 32. On the following day there is a further increase in the displacement of the heart; and there is still a slight cyanosis. Temperature 37.6; pulse 104; respiration 36. Puncture is made; and, after this, the patient feels much better.

No. 26 *b*: The patient took ill 3 hours before his admission to the hospital, with *a severe stitch in the chest, pallor and marked dyspnoea.* These symptoms were accompanied by *a protracted chill and some coughing.* On admission, he is not particularly exhausted but *rather pale, with a cyanotic hue.* Temperature 37.4. Pulse 112, regular, vigorous. Respiration 28. On the next day, the patient is yet a little cyanotic and slightly dyspnoeic, but the tension symptoms do not increase further.

No. 27 *b* (see fig. 28): The patient took ill the evening before his admission to the hospital, with severe pains in the left side of the chest and marked dyspnoea. On admission, he was *pale, perspiring and dyspnoeic.* Temperature 38. Pulse 120, slightly irregular. Respiration 36. The symptoms subsided considerably during the next two days, and no puncture was made.

No. 34. The patient took suddenly ill with pains in the chest, *dyspnoea and cyanosis.* Temperature normal. Pulse 140. Respiration 30. The following days there was a rise of the temperature, maximum 38.8, whereas the frequency of the pulse and respiration was decreasing every day; and when he was admitted to the hospital, 6 days after the onset, the symptoms had subsided a great deal.

The most pronounced example of tension pneumothorax is found in the case of No. 11.

This patient takes ill in the morning on the street, with severe pains in the right half of the chest and shortness of breath together with dry cough. He is immediately taken to the hospital: *cyanotic, congested, rather exhausted and suffering.* Temperature 37.5. Pulse 96. Respiration 40. Physical examination shows pneumothorax on the right side with displacement of the ictus to the anterior axillary line. The next morning the temperature is 37.7. Pulse 112. The liver is palpable just beneath the curvature. During the day there is an increase of the dyspnoea and the cyanosis. In the evening the temperature is 38.6. Pulse 100. Respiration 32. At 10 o'clock in the evening, *puncture* is performed, with evacuation of 1200 c. c. of air.

Initial intrapleural

pressure ..... + 13 cm. H_2O + 20 cm. H_2O.

After the puncture, which relieved the patient very much, the ictus was moved back to the papillary line. But a few days later, the displacement began again to increase, so that the ictus again moved out in the anterior axillary line. 9 days after, a puncture was again performed, this time with evacuation of 400 c. c. of air.

Initial intrapleural

pressure ..... + 2 cm. H_2O + 4 cm. H_2O.

After this, the patient kept on improving, and hardly 2 months after the onset of the lesion, only a very small amount of air remained in the lower part of the pleural cavity.

The characteristic of the tension pneumothorax is the exhausted and suffering condition of the patients, manifest by pains, dyspnoea, cyanosis, and collapse, sometimes ushered in with an initial chill.

The frequency of the respiration is from 30 to 40, or more. The pulse rate is often above 100, but it may be slower, even when the dyspnoea is marked. In most instances the temperature is elevated the first few days. Auscultation and X-ray examination show the typical picture of marked displacement pneumothorax. The intrapleural pressure is strongly positive.

In most of these cases the tension pneumothorax is established within the first 24 hours; but there are also cases in which the patients keep walking about and working with a milder degree of pneumothorax that gradually goes on to tension pneumothorax.

In some of the cases where tension pneumothorax is present immediately after the initial symptoms, the lesion culminates as early as in the first 24 hours of illness; but in other cases the tension symptoms are increasing further during the next 24 hours.

The prognosis of tension pneumothorax is always doubtful the first few days. Even when the pulse is vigorous and slow, the patient may die from sudden heart failure. However, this has not happened to any patient in my material.

After the first critical days are over, the respiration and the circulation become adjusted to the altered conditions, and the symptoms subside gradually.

The time it takes before all the air is absorbed from the pleural cavity will in most cases be 2 to 3 months.

CHRONIC PNEUMOTHORAX

In the preceding sections about the individual forms of pneumothorax it has been stated how long it takes before all the pneumothorax air is absorbed. Even in those cases where the patients come under treatment rather late, it is found that the air disappears within 3 months at the most when the patient is keeping quiet. In one case, No. 15, it took 8 months, however, before the last remnant of air had disappeared; but this was probably due — at least in part — to the fact that the patient did not keep his bed more than 9 days.

Yet, amongst my 51 patients there are 4 cases of spontaneous pneumothorax in which the disease was so extremely protracted that the patients were going about for years with chronic pneumothorax (No. 4, 6, 32, 35). These cases require some detailed mention.

Patient No. 4. This was a woman, aged 43, who had suddenly an attack of spontaneous pneumothorax on the right side with considerable displacement. She was in bed about 1 month, but in spite of the treatment her pneumothorax persisted unchanged for more than one year. 2 years after the onset, it had subsided, however.

During the past years there have probably been several relapses. On after-examination, 15 years after the first attack, the pneumothorax is all gone, and there are signs of a thickening of the pleura in the lower part of the right side of the chest.

Patient No. 6. At the age of 31 years, this woman had a sudden attack of spontaneous pneumothorax on the left side. The patient took this illness to be an attack of lumbago, and she went to a masseuse who gave her massage of the back. Later, as the pains persisted, the patient consulted a physician. X-ray examination, 2 months after the onset of the illness, showed a very marked degree of pneumothorax on the left side with displacement of the heart and mediastinum. At this time the intrapleural pressure measured $\div$ 6 cm. H_2O and $+$ 5 cm. H_2O.

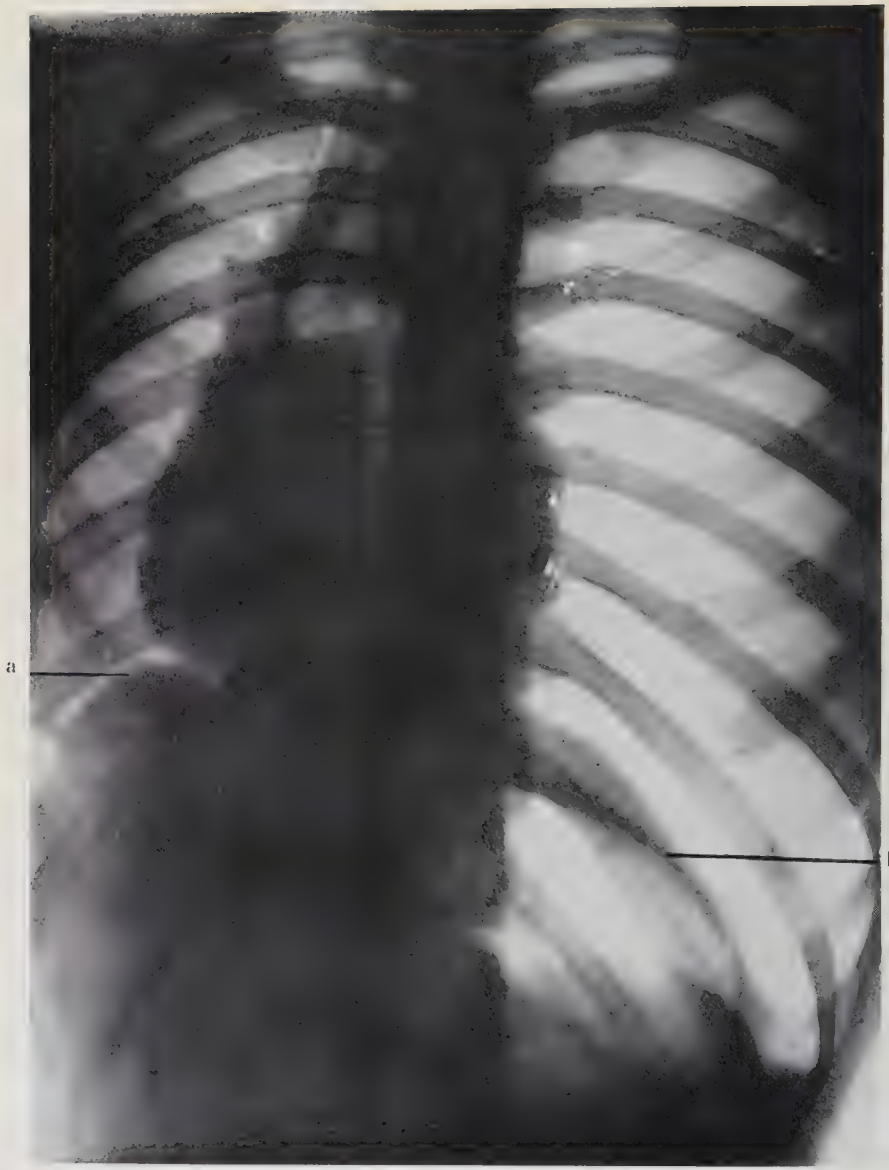

Fig. 23.

Chronic pneumothorax on the left side; plate taken over 1 year after the onset of illness. Pt. No. 6. Note the small exudate in the phrenocostal groove!

a: Right cupola of the diaphragm. b: Left cupola of the diaphragm; below this, the air-distended fundus of the stomach.

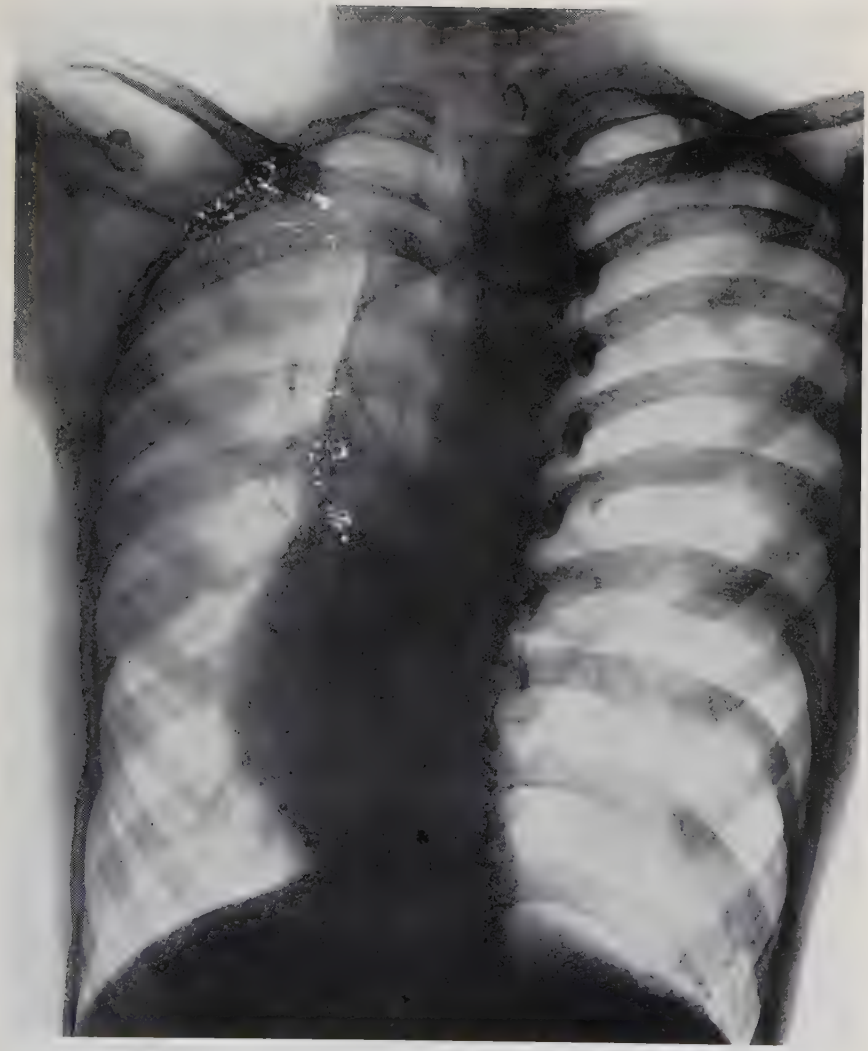

Fig. 24.

Chronic pneumothorax on the left side; probable onset ca. 14 years before this plate was taken. Pt. No. 32. Note the extreme displacement of the heart and the air-impression in the mediastinum! (cf. fig. 25 and 26.).

On examination 1 year later, the patient had still a marked degree of pneumothorax (see fig. 23).

This chronic spontaneous pneumothorax did not inconvenience the patient to any great extent, the only symptom she noticed being shortness of breath when working. She went through 2 parturitions, the first of these with forceps delivery. 7 years later, the affected left half of the chest, which had originally been distended, was now retracted and sunken, and the X-ray examination showed signs of diffuse opaqueness above and below, whereas the lung appeared expanded. The middle part of this lung field shows a clearance, probably a remnant of the pneumothorax. These upper and lower zones of opaqueness are probably due to thickening of the pleura and to the presence of adhesions, although the patient has never in the past years had any clinically demonstrable pleurisy, and on the whole, she has never been ill otherwise during the 14½ years which have now passed since the spontaneous pneumothorax first made its appearance.

Patient No. 35 is an example of pneumothorax discovered by chance. At the age of 12 years, this patient had pleurisy on the left side with effusion. At the age of 16, she had a sudden attack of pains in the right shoulder, accompanied by shortness of breath, but after staying in bed 4 days, she was again feeling perfectly well. After this, there have been now and then some transitory attacks of pains in the right shoulder, which were regarded as rheumatic pains.

When 22 years old, she was admitted to the hospital for some other lesion, and the question as to the cause of these pains was then taken up. It was now found that the patient had a small coat-formed spontaneous pneumothorax (see fig. 13). 3 weeks later, she had total pneumothorax with slight displacement (fig. 14) which went on to total pneumothorax with marked displacement (fig. 15).

This pneumothorax did now persist for about 1¾ year

after it had been discovered by chance, and then the lung began to expand without any special treatment. 9 month after the pneumothorax had been discovered, a few friction sounds could be made out over the lower part on the back. After-examination, ½ year after the expansion of the lung, shows a slight retraction of the right side, and the X-ray plate shows a slight dimness of the lung markings in the lower part of the right lung, suggesting a thickening of the pleura.

Patient No. 32 is likewise an instance of chronic spontaneous pneumothorax discovered by chance. When this patient was only 5—6 years old, the physician ascertained that his heart was in the right side of the chest; and as far back as he can remember, he has always felt the heart in the right side. When 19 years old, he was admitted to the hospital with symptoms of dyspepsia. Here it was ascertained that the dextrocardia of the patient was not due to any situs inversus, but to chronic pneumothorax on the left side with extreme displacement (see fig. 24).

After-examination, 2¾ years later, showed no change whatever in the extreme degree of total displacement pneumothorax. But on a subsequent after-examination, 5 ½ years after the pneumothorax had been discovered, it is found that the left lung now is partly expanded, and that the heart has moved back to its normal position. Now it is further ascertained that the affected lung is capable of partial function, as it occupies the upper and anterior part of the left pleural cavity. Back of the heart and extending up along the spinal column, however, there is a large elongated, walled-off, air space with a little fluid at the bottom, indicating that there still remains a large walled-off partial pneumothorax in the back of the pleural cavity. In this case, then, the spontaneous pneumothorax has probably persisted through 20 years, and now the lung has finally expanded partially, so that the original extreme displacement pneumothorax has now been reduced to partial pneumothorax (see fig. 25 and 26).

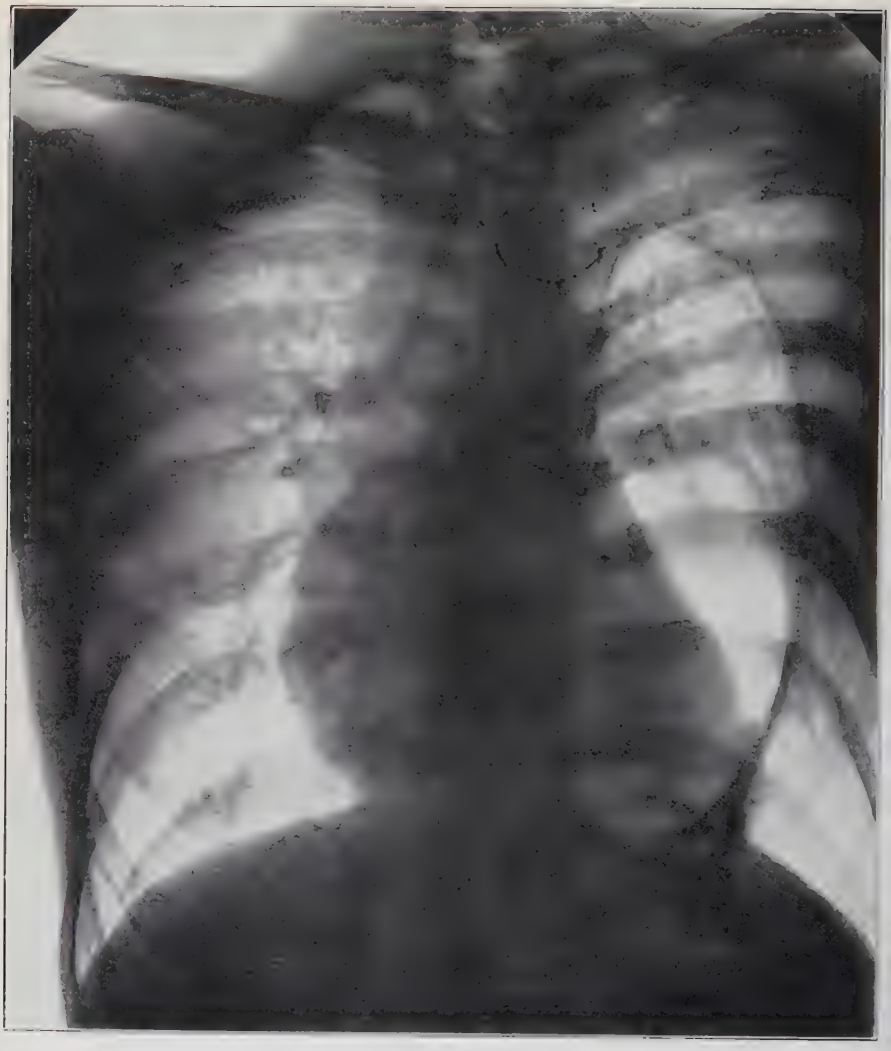

Fig. 25.

Same case as in fig. 24, taken 5½ years later. Pt. No. 32. Now the heart has almost returned to its normal place. The left lung is partly expanded. The medial half of the left lung field is the site of a large, sausage-shaped, clearing with a well-defined lateral arching border from C.IV to the diaphragm (Chronic partial pneumothorax).

Fig. 26.

Same case as fig. 24 and 25. Pt. No. 32. This plate is taken at the same time as fig. 25. Side-view, taken in standing position with the left side toward the plate. ×: Heart. Above the heart, the expanded left lung. Back of the heart, and passing up along the spinal column, is the clearing of the partial pneumothorax. ××: Small amount of fluid in the pneumothorax cavity.

These pneumothorax cases are instructive in several respects.

They have all persisted for years. In the 3 first-mentioned patients, the final result has been spontaneous recovery, the lung having expanded again. As remains of the pneumothorax, these 3 patients show a more or less pronounced degree of pleural thickening; in one of them (No. 6) there is a considerable retraction of the affected side of the chest wall, and in another patient (No. 35) there is a lesser degree of retraction. The last patient, No. 32, on the other hand, presents a case of spontaneous pneumothorax which developed in childhood and has now persisted about 20 years; yet, within the last few years, there has been a spontaneous partial expansion of the lung, and this instance of total displacement pneumothorax has now (September, 1931) regressed to a state of partial pneumothorax.

The two last mentioned cases of pneumothorax have been discovered accidentally in patients who were admitted to the hospital with other complaints.

In the case of No. 35, it is peculiar that one may follow on the X-ray plates how the pneumothorax progresses quite gradually through several weeks. In this case the attack of pneumothorax has been a relapse. But the insidious, almost unnoticeable, manner in which the pneumothorax of this patient develops suggests very strongly that most of the rare cases of pneumothorax without any initial stage whatever have developed in the same gradual manner. As far as I have been able to find out, such a slow development of spontaneous pneumothorax has not been described in the literature before.

In the last-mentioned case, No. 32, the past history of the patient is altogether lacking in data suggestive of an acute initial stage of the pneumothorax, and this condition was not diagnosed till many years after its onset.

In the two first-mentioned cases, on the other hand, we find an acute initial stage. But, in spite of rational treatment, the course of these cases has been so protracted

that the patients have been going about for years with their pneumothorax. The cause why such cases of spontaneous pneumothorax become chronic must naturally rest with some especially poor conditions for the healing of the rupture of the valve vesicle, but nothing may as yet be said as to the nature of these obstacles to the healing of the rupture.

In the literature only a very few cases of chronic spontaneous pneumothorax have been reported so far. These have been published by Adams, Whitney, Bittorf, Hayashi, Schreiber, Magnus-Levy, Brunner, Als and le Wald.

Some of these cases have been discovered quite accidentally in patients who have never been troubled by their pneumothorax.

This applies to the patient of Emil Als (case No. IV) who was admitted to the surgical department with the chief complaint of goitre, on which occasion the physical examination revealed the presence of a valve pneumothorax which has presumably set in about one year before. It this case, recovery was successfully accomplished by means of aspirations.

Bittorf's patient differs from the other patients of this group in being very dyspnoeic. This patient was a woman, 48 years old, with chronic pneumothorax on the left side, in whom the atmospheric pressure within the pleural cavity was the sign of a patent fistula. The heart was markedly displaced to the right side. According to her past history, the lesion set in when she was 23 years old, and it had always troubled her a great deal, so that throughout the past 25 years she had been unable to do any house-work.

Hayashi has reported a case of chronic spontaneous pneumothorax in which the date of onset was unknown, and where the lesion was first revealed on autopsy. This case has been mentioned before, on p. 20.

Adams (1886) observed a case of chronic spontaneous pneumothorax which was not recognised till 2 years after its onset. After persisting 5 years, it subsided.

Whitney described, in 1899, a case of pneumothorax that had lasted 9 years.

Schreiber (1917) has reported the case of a patient who was asking his advice for pains and tenderness of the epigastrium which had troubled him several years. Physical examination revealed the presence of a hitherto unnoticed pneumothorax on the left side. This was treated successfully with aspirations, and as the patient recovered from the pneumothorax, his epigastric pains disappeared.

Magnus Levy (1918) has described a case of chronic pneumothorax which presumably had set in during childhood, persisting about 20 years.

Brunner's 2 cases have already been mentioned, on p. 21—22.

Le Wald[3]) (1928) has reported one case in which it took 1 year before the lung began to expand, and another case — of an elderly woman — in which the pneumothorax was still persisting 11 years after its onset.

As a rule, chronic spontaneous pneumothorax is associated with a very marked displacement of the heart, and this may go on to such extreme degrees as are hardly ever encountered in the acute cases of severe tension pneumothorax (cf. fig. 23 and 24). This is due to the fact that even a slight difference in the intrapleural pressure of the two pleural cavities will in the length of time effectuate even a greater displacement than may be produced by a considerable overpressure within a short time.

In some of these chronic cases it is the question of a patent pneumothorax with no overpressure whatever in the pleural cavity. Still, as the intrapleural pressure on the healthy side is negative, the difference between this negative pressure and the atmospheric pressure in the pneumothorax cavity is sufficient to produce a marked displacement.

This was the case in the above-mentioned patient of

Bittorf, and this fact is also plainly evident from fig. 23 (No. 6). This X-ray plate illustrates very well the great displacement that had resulted, although the intrapleural pressure in this case kept varying around the atmospheric pressure, the pressure reading showing: ÷ 6 and + 5 cm. H_2O.

In some cases, then, the chronic pneumothorax is a patent pneumothorax with atmospheric intrapleural pressure, while in other cases it is a chronic valve pneumothorax with intrapleural overpressure.

The latter condition was met with in the patient of Emil Als, in which case the pressure reading showed: ÷ ½ and + 8 cm. H_2O.

The characteristic feature of the chronic pneumothorax is the very contrast between the marked pneumothorax with the great displacement and the comfort of the patient, who only feels a little short of breath when working. The only exceptions from this rule are the two cases reported by Bittorf and by Brunner.

Some of these chronic cases set in acutely, others — as No. 35 — develop quite gradually. But local conditions of unknown nature at the site of the perforation bring about that these cases are sometimes persisting, all rational treatment notwithstanding, because the perforation — be it ever so small — fails to close.

Even after a refractory persistence through a number of years, however, most of these cases will finally show an expansion of the affected lung. But there are a very few cases in which there is no tendency whatever to recovery, so that the pneumothorax of the patient persists for dozens of years.

The treatment of these cases will be mentioned later.

HÆMOPNEUMOTHORAX

In the chapter of etiology, mention is made of two fatal cases of hæmopneumothorax reported by Pitt (p. 20.) and by Fischer (p. 21.). In Pitt's case the autopsy revealed a rupture of a solitary vesicle of emphysematous origin, at the apex of the lung, and a tear of a small adhesion wherefrom the hæmorrhage was thought to come. In Fischer's case the autopsy showed a ruptured scar-tissue vesicle at the apex with fresh coagula at the site of the rupture, so that here it was reasonable to ascribe the hæmorrhage to the rupture of a blood vessel in the scar-tissue vesicle.

My material comprises 2 patients in whom the pneumothorax was associated with hæmorrhage into the pleural cavity.

In one of these cases, No. 36, the hæmorrhage was only slight (see fig. 21 and 22), and the clinical appearance of th case had entirely the character of tension pneumothorax. It is therefore described as such a case, on p. 77. There was a slight rise of the temperature the first couple of weeks.

But in the other case, No. 23, we have a pronounced example of hæmopneumothorax.

This patient was a young man, aged 20, with a past history of good health, who was *suddenly taken ill one morning with pains in the left side of the chest* and in the back. The pains were aggravated on deep respiration.

He felt bad during the day, and yet he stayed up all day. *In the evening — and not until then — he was dyspnoeic; the pains in the side were getting worse; and the temperature rose to about 39. He was now very exhausted, and he had a severe attack of collapse.* He was admitted to the hospital 3 days after the onset of illness. He was then anæmic and dyspnoeic. Temperature 38.8. Pulse 100. Respiration 28. Auscultation and X-ray examination showed pneumothorax on the left side with a fluid in the pleural cavity, reaching on the back to costa VI. The heart was entirely displaced to the right side. On explorative

puncture the fluid in the pleural cavity turned out to be blood. Hæmoglobin (Sahli), 6 days after the onset: 53 %. 15 days after the admission, puncture was made, and 400 c. c. of bloody fluid was evacuated. There was some rise of the temperature the first 2 weeks. The patient was discharged as well, and he has been perfectly well the past 4¼ years.

This case, then, is an example of hæmopneumothorax where the hæmorrhage stops spontaneously, and this saves the life of the patient.

Beside the cases reported by Pitt and by Fischer, the literature contains up to the present only 7 cases of hæmopneumothorax. These are as follows:

Rolleston (1900) has reported a case of fatal hæmopneumothorax, in which the autopsy failed to clear up the etiology:

A 24-years-old man, with a past history of good health (apart from some dyspeptic trouble), had a sudden attack of severe pains in the right hypochondric region, radiating out toward the right shoulder and down toward the umbilicus. 24 hours later, the patient was brought to the hospital in a condition of extreme collapse and marked anæmia (pulse 160, irregular). The abdomen was tender on palpation, rigid. The *diagnosis* was made: *Perforation of Duodenal Ulcer with Diffuse Peritonitis.* No operation was performed, as the patient was considered moribund. Next day, the patient had improved a little. The pulse was 144, the respiration 44, and now there were marked signs of right-sided spontaneous pneumothorax. Puncture was performed the following day, with evacuation of blood and air. One week after the onset of illness, the patient had a severe attack of bronchitis in the other lung, and he died. *Autopsy:* The right pleural cavity contained about 2 liters of dark fluid blood and some coagula. In spite of careful examination of the lungs with insufflation under water, no perforation could be demonstrated. The lungs were perfectly normal; there were no adhesions.

Boland (1900): A 34-years-old man, with a past history of good health, woke up in the night with severe pains in the right side of the chest. The next morning he showed marked signs of pneumothorax. He improved somewhat, but as there now developed signs of fluid in the pleural cavity, puncture was performed 3 weeks after the onset of illness, with aspiration of about 1 liter dark blood. As the amount of fluid in the chest was again increasing, the aspiration was repeated 15 days later, with evacuation of ca. 1200 c. c. almost pure liquid blood. The patient recovered. Boland takes this to be a case of „idiopathic hæmopneumothorax" due to rupture of old adhesions.

R. B. Ness & G. A. Allen (1910): A 31-years-old man had a sudden attack of pains in the left side and in the abdomen; at the same time he was feeling rather short of breath. He stayed at home, quietly, the next couple of days; the following day he went to work again. One week after the onset of illness, while on his way to work, he had a sudden attack of very severe pains in the left side of the chest and in the left shoulder. He came near fainting, and yet he managed to get home by tramcar. Physical examination showed the presence of hæmopneumothorax on the left side. From the left pleural cavity, about 150 c. c. of serosanguinous fluid was evacuated together with some air. There was succusio Hippocratis. The ictus was seen and felt to the right of the sternum, and there was also a distinct displacement of the liver and spleen. The temperature was elevated moderately during the first week, whereafter it was normal. The patient made a complete recovery in 6 weeks. He gave a weakly positive Pirquet reaction. X-ray examination was not made.

Rudolf Kiær (1923) has reported a fatal case of hæmopneumothorax, in which the autopsy — as in Rolleston's case — failed to give any information of the etiology:

A 30-years-old man, who had been well before, was suddenly taken ill with pains in the back of the neck and between the shoulder-blades. Two days later, when Dr.

Kiær was called, the patient had also been dyspnoeic a few hours, and the diagnosis was made: Pneumothorax with considerable Pleural Effusion on the left side. Next morning there was increasing dyspnoea and anæmia. Chief physician v. Thun was consulted; and puncture was performed, with evacuation of air and a considerable amount of blood. The following night, the patient died from anæmia. *Autopsy* (performed under the best conditions possible) failed to reveal any pathological changes in the lung. The pleural surface was smooth and glistening throughout; there was no trace of any adhesions, of old or recent origin; and it was impossible to find any break in the continuity of the surface.

G. A. Allan (1925) has further published 2 other cases of hæmopneumothorax, in which the bleeding ceased spontaneously:

1) Male, 20 years old. One day the patient felt uncomfortable, with slight pains in the left side of the chest that kept him from working. At 3 o'clock the next morning he had suddenly a severe stitch in the back, and his physician, who was called immediately, ascertained the presence of spontaneous pneumothorax on the left side. The temperature was normal; the pulse was 80. At 9 in the morning the patient collapsed suddenly, and his condition was critical all day long. On the next day the physical examination showed pneumothorax with dullness over the back and succussion. An explorative puncture yielded pure blood. Temperature normal. Pulse 136. Respiration 48. The patient improved gradually, and the temperature was perfectly normal throughout his illness except on one day. After-examination, 9 months later, showed only a very slight suppression of the breath-sounds over the left side of the back. X-ray examination showed a small tuberculous infiltration in the lower part of the left lung, and scattered minute infiltrations in the right lung.

2) Male, 38 years old. One morning, at 6 o'clock, the patient felt some pains under the right shoulder-blade.

After breakfast he was feeling better, but several times in the day he had the same pains, which subsided when he was sitting down. In the evening he had suddenly a very severe stitch in the right side of the chest, accompanied by fainting spells and exhaustion bordering on collapse.

Physical examination showed pneumothorax on the right side. The heart was displaced to the left. The upper border of the liver dullness was somewhat lower than normal, and there was *rigidity of the upper right quarter of the abdomen.* There were dullness over the back and succussion sounds.

Explorative puncture gave pure blood. The patient improved rapidly. The temperature was normal throughout his illness, with the exception of one day. The X-ray picture taken 4 months later was suggestive of tuberculosis in the upper half of the right lung.

Although these two cases on X-ray examination showed minor tuberculous changes, and they are thus falling outside my definition of spontaneous pneumothorax in the apparently healthy, in their clinical features they are still approaching the other cases of hæmopneumothorax so much, that it is instructive, I think, to mention them for the sake of comparison.

Hurxthal (1928) has described "an unusual case of spontaneous idiopathic hæmopneumothorax with certain features resembling an acute surgical abdomen":

Male, 29 years old. Past history of good health, apart from dyspeptic symptoms the last 2—3 years. One day he had suddenly a violent stitch under the right scapula. He went to bed, and shortly after he began to be short of breath. In the following hours there was an increase of the dyspnoea and the pains in the right side, besides pallor and abdominal pains. He was therefore brought to the hospital. On admission, the patient was pale and sweating. The pulse was thready, over 120. Blood pressure 80/70. Physical examination showed tympanism and suppressed respiration over the right front. Liver dullness absent. The

heart displaced about 3 cm. to the left. Variable dullness over the back. *The abdomen was tense and tender, especially in the epigastrium and in the right upper quadrant, suggesting the possibility of an acute lesion in the abdomen,* but no such diagnosis was made. On the following day an explorative puncture yielded pure blood; and on the next day, aspiration was made of 2400 c. c. of dark-looking uncoagulated blood, which was replaced with air through insufflation. The temperature was elevated only during the first 3—4 days. 11 days later, the puncture was repeated — with evacuation of 1 liter blood. The patient improved rapidly, and he got up 10 days after the onset of illness. X-ray examination showed no sign of tuberculosis.

Surveying the aforementioned cases of spontaneous hæmopneumothorax, the result is:

Spontaneous cessation of the bleeding in 6 cases (Boland, Ness & Allan, Allan (2 cases), Hurxthal, and the author).

Death in 4 cases (Pitt, Rolleston, Fischer, Kiær). In 3 of these cases the patients bled to death (Pitt, Fischer, Kiær);

in 1 case (Rolleston) the patient died with a complication of bronchitis in the other lung, in connection with the hæmorrhage into the pneumothorax cavity.

It is indeed a very peculiar fact that an oozing bleeding from such a small blood vessel in a small superficial vesicle or in an adhesion may give rise to such a serious hæmorrhage in patients who are not hæmophilic.

Another peculiar finding is the fact that the blood in the pleural cavity shows such little tendency to coagulation.

The *characteristics of spontaneous hæmopneumothorax* may then be summed up as follows:

When the lung ruptures, the patient feels at first some rupture pain and, sometimes, a moderate shortness of breath. During the following hours the patient does not feel quite well, but not particularly bad either.

The signs of internal hæmorrhage, usually sudden, do not appear untill several hours after the rupture: Fainting spells, collapse, profuse sweating, pallor, tachycardia, and fall of the blood pressure. These symptoms are accompanied by pains and by dyspnoea, resulting from the displacement of the heart and mediastinum through the accumulation of blood and air in the pleural cavity.

Sometimes vomiting sets in, and the clinical picture of the disease is complicated with a marked tenderness of the epigastrium and, in right-sided hæmopneumothorax, with tenderness and muscular rigidity in the upper right quadrant of the abdomen, so that it may have a deceptive resemblance to the clinical features encountered in perforation of a gastric ulcer (as in the cases of Rolleston, Fischer, Allan, and Hurxthal).

The temperature is increased in some cases, but it may also be normal, and in the stage of collapse it may even be subnormal.

This lesion is always very serious, involving the danger of death from internal hæmorrhage.

The treatment will be mentioned later.

RECURRENT PNEUMOTHORAX

A peculiar feature of spontaneous pneumothorax in apparently healthy individuals is its tendency to recurrence. This is easy to understand in view of the fact that the lesion is due to the rupture of a valve vesicle. Once the wall of such a vesicle has become so thin an stretched that it ruptures, the patient has his first attack of pneumothorax. After the small perforation of the valve vesicle has healed, the patient, of course, has still his thin-walled valve vesicle, which naturally keeps on involving the risk that it may rupture anew. Yet there is also the possibility that the recurrence of the pneumothorax may be due to the rupture of another valve vesicle, for, as mentioned in the section of etiology, the scar-tissue vesicles are often multiple.

In most of these cases the recurrence appears on the same side as the first attack, but, in rare instances, rupture takes place in the other lung. The later form — the alternating pneumothorax — is mentioned in the following section.

Nikolsky as well as Ljungdahl find that amongst the cases reported in the literature, relapse have been described in about 10 per cent.

In my 51 patients, 7 have had relapses (No. 8, 12, 26, 38, 41, 42, 46); and in addition there is 1 patient with alternating pneumothorax (No. 27).

In the first of these patients, No. 8, the first attack, in 1915, was very mild. 2 years later the patient was taken ill with an ailment that was regarded as dry pleurisy, but it may have been a relapse of pneumothorax. Finally, in 1924 — 8¾ years after the first attack — she had an attack of fully developed spontaneous pneumothorax.

No. 12 had 2 attacks of tension pneumothorax, 8 months apart.

No. 26 had 2 attacks of tension pneumothorax, 7 months apart and subsequently, perhaps, 2 mild attacks of spontaneous pneumothorax.

No. 38 had 2 attacks of coat-formed pneumothorax, 2½ years apart.

No. 41 had 3 attacks of total pneumothorax, within 3½ years. Between the first two attacks there was an interval of 2 years.

No. 42 had 3 attacks of total pneumothorax, within 4 years. There was an interval of 2 years between the first and second attacks, and a similar interval between the second and third.

No. 46 had 2 attacks of partial pneumothorax, 7 months apart.

Beside these 7 patients in whom the relapses were observed with certainty, there are 3 other cases in which the anamnesis suggests very strongly that the patients have had previous non-recognised mild attacks of spontaneous pneumothorax before they had a marked attack (No. 24, 35, 44).

No. 24 has probably had 3 preceding mild attacks within a period of 4 years.

No. 35 has probably had several mild attacks in a period of 6 years.

No. 44 has probably had one attack 2 years before the one recorded here, and one or two after this.

Possibly No. 15 and No. 40 have also had preceding mild attacks.

Finally, No. 4 has probably had several relapses, but sufficient data of this are lacking.

So, I think, it will be more correct to give *the frequency of recurrence as about 20 per cent of all the cases,* for it is by no means a rare instance that the history of the patient brings information suggestive of mild attacks which have not been diagnosed.

It is impossible to say which patients are liable to have relapse. The patients with relapses have been treated just like the other patients, and they have been kept in bed just as long as the others, so their relapses cannot be attributed to inadequate confinement to bed. It is further

impossible to say how an eventual relapse will turn out. It may resemble the first attack; it may also be milder, or more severe.

In No. 8 there was a period of 8¾ years between the first and the second attacks. Such a long interval is exceptional, however, as most of the relapses occur within one year after the first attack.

This is evident from the following survey of the *literature* supplemented with my own cases:

Bull [1, 2]: 4 attacks within 2 years (4 months between the first two attacks).

Flint[2]*:* 2 attacks, 3 months apart.

Gabb: 4 attacks in 13 years (4 years between the first two attacks).

West[3]*:* 3 attacks in 4 years (8 months between the first two).

Finny: 2 attacks, 4 months apart.

Weber[1]*:* 2 attacks, 2 years apart.

Jochmann: 2 attacks, one right after the other.

Sale: 11 attacks in 6 years (1 year between the first two).

Hamilton: 2 attacks, 1 year apart.

Cummer: 2 attacks, 6 months apart.

Huet: 2 attacks, 1 year apart.

Dahlstedt & Haeger (case II): 2 attacks, one right after the other.

Deutsch: 7 attacks in 3 years (no exact dato on the intervals).

Koelensmid (case I): 3 attacks in 1½ years (16 months between the first two).

Duhot: 2 attacks, 6 years apart.

Watson & Robertson (case II): 2 attacks, 8 months apart.

Laufer: 2 attacks, 5 years apart.

My material: No. 8: 2 attacks, 8¾ years apart.

No. 12: 2 attacks, 8 months apart.

No. 26: 4 attacks in 3 years (7 months between the first two).

No. 38: 2 attacks, 2½ years apart.

No. 41: 3 attacks in 3½ years (2 years between the first two).

No. 42: 3 attacks in 4 years, at intervals of 2 years.

No. 46: 2 attacks, 7 months apart.

In these 24 patients, then, we find:

2	attacks	in.....	15	patients,
3	„	-	4	„
4	„	-	3	„
7	„	-	1	patient,
11	„	-	1	„ .

In 15 out of these 24 patients the relapse came within 1 year.

ALTERNATING PNEUMOTHORAX

In very rare instances one may meet with a patient who, after recovering from spontaneous pneumothorax on one side, has a new attack of pneumothorax on the other side — some time, short or long, after the first attack. The explanation of such a case must be that the patient has valve vesicles in both lungs (cf. p. 21—22).

In my material there is one patient (No. 27) with such alternating pneumothorax.

This patient had first an attack of right-sided total pneumothorax without displacement (see fig. 27). 7 months later he had an attack of tension pneumothorax on the left side (see fig. 28).

Goodhart is the first to report a case of this kind, his patient having first an attack of pneumothorax on the right side and then, about 1 year later, a new attack on

the left side. Grenier's patient had one attack on the left side, followed by two attacks on the right side. Hamman's patient (case I) had first pneumothorax on the right side and then, about 1 year later, 2 attacks of pneumothorax on the left side (cf. p. 114.). Sior has described a case of alternating pneumothorax in a child, 11 years old. Hawes has reported a case with several attacks on either side during a period of 10 years. Morrison has published a case of alternating pneumothorax in which the attacks came in rapid succession; the same applies to a case reported by le Wald[2]. In Atwood's patient, on the other hand, the attacks were 23 years apart; at the age of 37 this patient had pneumothorax on the left side, and when 60 years old he had an attack of pneumothorax on the right side.

Watson & Robertson have described two very pronounced cases of alternating relapsing pneumothorax. One patient (case I) was a physician, aged 32, who within ca. 10 years had 7 well established attacks of pneumothorax and several minor attacks, some on the right side, some on the left. The attacks were brought on by moderate exertion — for instance, carrying a heavy suitcase or lifting a patient. After this period, about 10 years, of recurrent alternating pneumothorax, the attacks left off, so that there were no attacks between 1925 and 1928, when the case was reported.

The other patient (case III) had in a couple of years no less than 14 attacks of pneumothorax, now on one side, now on the other; and once he had a bilateral pneumothorax — extensive pneumothorax on the right side and a coinciding partial pneumothorax on the left side (at the apex).

Taking the few reports of alternating spontaneous pneumothorax, it is found that in most of these cases the second attack sets in some months, or about 1 year, after the first attack.

Fig. 27.
Alternating pneumothorax. Attack on the right side. Pt. No. 27.
a: Right lung.

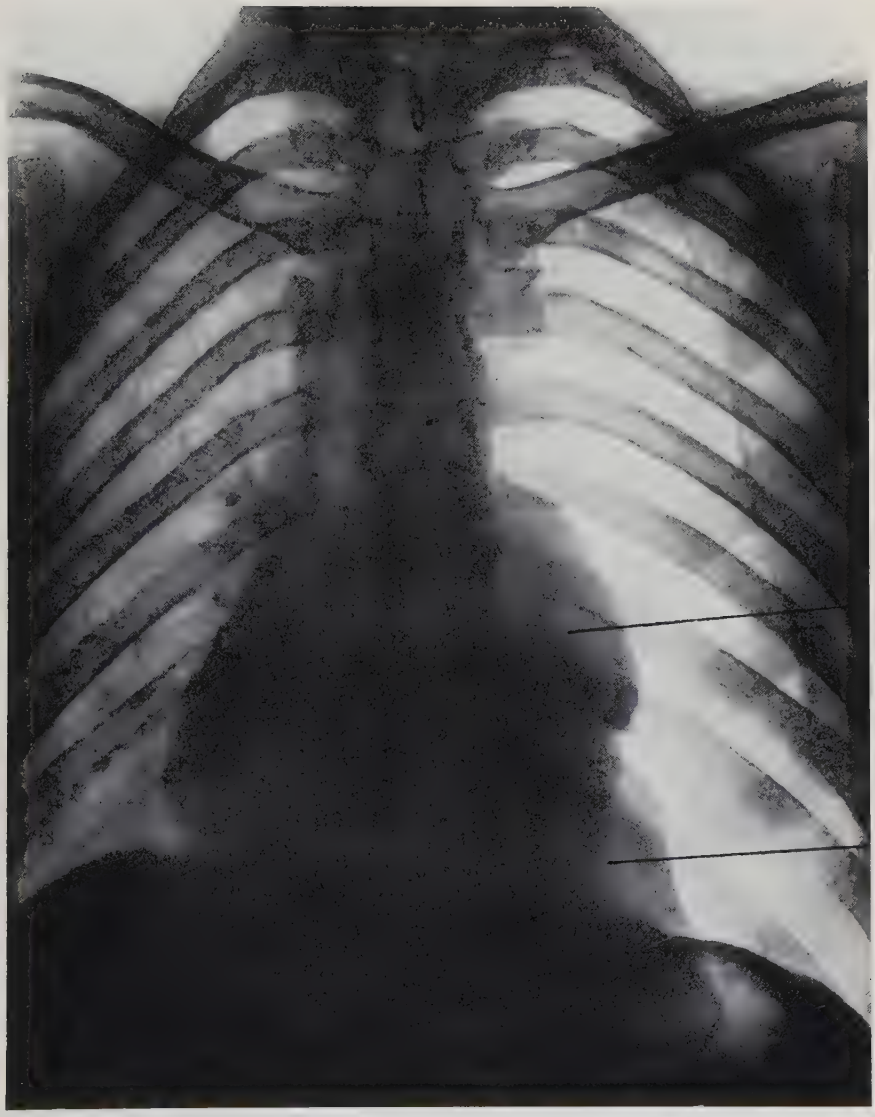

Fig. 28.
Same patient, No. 27, as in fig. 27. Attack on the left side, 7 months after fig. 27.
a: Upper lobe of the left lung. b: Lower lobe.

This is confirmed by the most recent case of this condition, reported by Mills; and my case shows the same relation.

BILATERAL SPONTANEOUS PNEUMOTHORAX

The alternating pneumothorax and the bilateral pneumothorax, a much more serious and truly critical condition, have the same etiological basis: in both of these conditions, the patients have valve vesicles in both lungs.

The bilateral spontaneous pneumothorax does not set in at the same time on both sides. To begin with the lesion is always unilateral, but as it overtaxes the functional capacity of the other lung, the forced respiration is subsequently giving rise to a rupture of the other lung too, whereby the patient is *immediately* in danger of suffocation.

In severe degrees of chronic substantial emphysema of the lungs, a few instances of bilateral spontaneous pneumothorax have been described, all terminating in death (Massini & Schönberg, Emerson & Beeler, Meyer). An exception from this is Faschingbauer's case of bilateral partial pneumothorax (see p. 114.).

Bilateral spontaneous pneumothorax in the apparently healthy is not represented in my material. Beside the aforementioned case of Watson & Robertson (case III), the literature contains only 2 cases of this lesion, reported by Hayashi and by Elte.

Hayashi's case has been mentioned in detail in the chapter of etiology (p. 21.).

In 1928, Elte described this interesting case:

Male, aged 19. On April 19, the patient had suddenly a stitch in the right side of the chest, accompanied by shortness of breath. The next day he had to quit working. The following two days, the dyspnoea and the pains in the chest were aggravated. On April 23, he was admitted to the hospital, showing right-sided tension pneumothorax

with displacement of the heart to the left, marked dyspnoea and cyanosis. Pulse 108. After the physical examination, morphine was given, and the patient began to feel less dyspnoeic. But ¾ hour later, he suddenly got very ill with extreme cyanosis and dyspnoea. So the nurse called Dr. Elte immediately. Shortly after, the patient fell back in his bed, unconscious, with irregular respiration (long pauses between periods of breathing). The tongue was protruding, foam was issuing from the mouth, and the eyeballs were turned upwards. A hasty examination showed that pneumothorax had set in on the left side too. Puncture was performed immediately on the right side, and the cannula was connected with a suction pump by means of rubber tubing.

During the first few minutes after the aspiration had been started, the patient was still on the point of dying. But then his breathing began to be more frequent and regular. Yet, even after 20 minutes' aspiration, the patient was still unconscious. So puncture was now performed on the left side too, and both cannulas were connected with an electrical suction pump.

Shortly after this bilateral aspiration had been established, the respiration became normal, and the cyanosis disappeared; the patient opened his eyes, and he was again conscious. Now some faint breath-sounds could be made out over the upper part of the chest. The pointed cannulas were replaced with blunt ones, and the suction pump was put out of function.

Later in the day the aspiration had to be repeated. After this, both cannulas were left in connection with the suction apparatus, which was not removed till one week later. One of the cannulas was even left in situ a few days longer.

X-ray plates, taken 11 days after the onset of illness, showed a bilateral pneumothorax, though not particularly extensive on either side. The lungs were expanding gradually. But in August — that is, about 4 months after the

first attack — there was again pneumothorax on the left side; and this persisted till the beginning of October, whereafter the patient was discharged as well. No sign of tuberculosis could be made out.

Elte emphasizes that the puncture and aspirations were first carried out on that side were the pneumothorax had been present the longest, because there was here some chance of the perforation having shut close; and he considers it important that the aspiration is limited to what is required for relieving the patient of his difficulties of breathing.

In both of these two cases of spontaneous pneumothorax in apparently healthy individuals there was a considerable period between the two ruptures. In Hayashi's case the spontaneous pneumothorax was unilateral for 3 weeks before the rupture took place on the other side. In Elte's patient the pneumothorax was unilateral for 4 days before it became bilateral.

Elte's case is a beautiful illustration of the fact that a moribund patient of this kind may be saved by timely and well-calculated aspiration of both pleural cavities.

PNEUMOTHORAX ALTERNATING WITH PNEUMOPERICARDIUM

In the literature there are 2 instances of spontaneous pneumothorax and pneumopericardium occurring in the same patient at different times.

Lundie (1891) has described the following case:

Male, aged 40. On April 28, 1890, the patient had suddenly an attack of severe pains under the left shoulder-blade and marked difficulty of breathing. He was found to have *pneumothorax on the left side*. The temperature was normal. 2 weeks later the patient was well enough to get up. On June 14, he had suddenly a new attack of similar pains. But this time his lesion was found to be

pneumopericardium. And this time, too, the temperature was normal. One month later, the patient was well again; and the after-examination, 1 year later, did not show any abnormality.

Ljungdahl[1, 2] (1918) has reported the following case:

Male aged 17. On October 3, 1912, the patient had suddenly a stitch in the left side of the chest, accompanied by dyspnoea. On auscultation, the same day, pericardial friction sounds were heard distinctly, but the next day they were gone, whereafter the presence of *pneumopericardium* was ascertained conclusively. There was no pericardial effusion; and the temperature was normal. 12 days after the onset, the patient was well again. He was now in good health for about 1½ years, till June 6, 1914, when he suddenly had an attack of *right-sided spontaneous pneumothorax* with cyanosis, dyspnoea and displacement of the heart to the left. The temperature was normal — except on one day when it rose to 38. The patient recovered in 3 weeks. After-examination, 3 years later, showed no abnormalities, and the patient had been perfectly well in the meantine.

Lundie claims that this condition is to be explained as resulting from a rupture of the lung at a place where an old pleurisy has involved the pleural surface of the pericardial sac; and Ljungdahl advances the hypothesis that this may be a question of the results from a benign inflammatory process originating from tuberculous mediastinal glands.

After the subsequent investigations published by Hayashi and Fischer on scar-tissue vesicles it seems most reasonable to think that these patients have some scar-tissue vesicles at a place where the right, or the left, visceral pleura is firmly adherent to — or fused with — the pleural covering of the pericardium, these vesicles being located so as to imply the possibility of rupturing into the pericardium and into the free pleural cavity.

PNEUMOTHORAX WITH SUBCUTANEOUS EMPHYSEMA

If a marked increase of the intrapulmonary pressure — e. g., through a violent coughing spell — gives rise to an alveolar rupture, this may result in a subcutaneous emphysema, appearing first on the neck and then extending further out under the skin. This is owing to the circumstances that under the pressure from the forced respiration or from the coughing spell, the air is pressed from the site of rupture along the interlobular and peribronchial connective tissue into the mediastinum, wherefrom it passes on to the neck region.

In adults such an alveolar rupture, leading on to subcutaneous emphysema is a very rare phenomenon.

It may occur during parturition, resulting from the labour pains in the stage of expulsion of the child (Lauritzen), and further during strenuous defæcation or lifting heavy weights (Sinnhuber), or under an attack of asthma (Morawitz).

In these cases, thus, there is no rupture of the pleura — which, in fact, is more resistant than the alveolar wall — and, consequently, pneumothorax does not result.

But in infants and young children, whose lung tissue is less resistant, such an alveolar rupture with subcutaneous emphysema does not appear to be so rare as in adults. This lesion may be met with in severe attacks of whooping-cough (Aaser, Reyher), in bronchopneumonia (Moncrieff, Gielczynski), and also in other infectious diseases with involvement of the lungs — e. g., measles.

In Gielczynski's case there was in addition an attack of pneumothorax, resulting from a subsequent rupture of the pleura.

During an epidemic of influenza, Berkely & Coffen have observed 9 cases of bronchopneumonia complicated with subcutaneous emphysema; 2 of these were further complicated with spontaneous pneumothorax.

In my material there are 2 cases of spontaneous pneumothorax with subcutaneous emphysema.

One of these, which has been mentioned already (p. 67.), was a rather slight attack of partial pneumothorax on the right side, accompanied by a slight and transitory degree of subcutaneous emphysema above and below the right clavicle. 8¾ years later, the patient had an attack of total pneumothorax on the same side.

In this case it is reasonable to assume the presence of some scar-tissue vesicles, adherent in part to the mediastinum, so that they may rupture into the mediastinum and give rise to subcutaneous emphysema as well as into the free pleural cavity and produce pneumothorax.

The other case, No. 48, shows quite a different clinical picture and a very serious one. As its history ought to be read in extenso, it will not be abstracted here. The reader is referred to the case record and to fig. 29.

There can be no doubt, that in this case the mediastinal emphysema is the primary lesion, and that the right-sided spontaneous pneumothorax has appeared a few hours later.

On account of the chronological sequence of the phenomena, and because the patient is a child of only 5 years, it is not very likely that the disease in this case is due to rupture of a valve vesicle. There has probably been a primary alveolar rupture, resulting from a coughing spell, with secondary pneumothorax.

Yet I have taken this case along with my other cases, partly because such instances are extremely rare, and partly on account of the beautiful result that was obtained in this case with the treatment, saving the life of the moribund child.

But, even leaving out this last case, No. 8 does yet illustrate that *spontaneous pneumothorax in the apparently healthy may be associated with subcutaneous emphysema.*

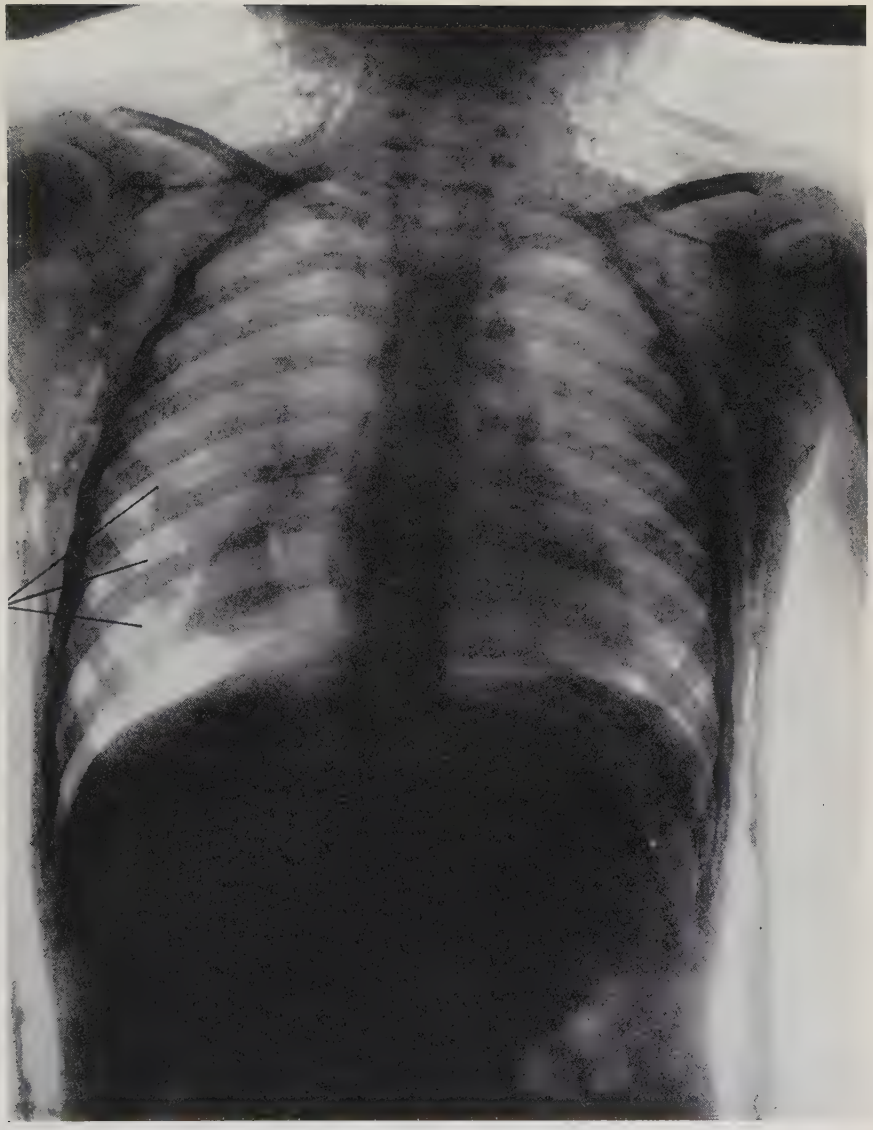

Fig. 29.
Right-sided pneumothorax with subcutaneous emphysema in child, 5 years old. Pt. No. 48.
Note the enormous accumulations of air under the skin of the neck, axilla and chest!
a: Surface of the right lung.

DIAGNOSIS

When one is facing a case of spontaneous pneumothorax in an apparently healthy person, the diagnosis will as a rule be rather difficult. In the first place, it may be hard to realise that the patient has pneumothorax at all; and secondly, one may question whether it is *this* particular form of pneumothorax one is dealing with.

The only symptom that is constantly present, is the suppressed respiration, but sometimes this is not very marked.

In partial and in coat-formed pneumothorax the whole picture of the disease is so little suggestive, that neither the history of the patient nor the auscultation may lead to more than a provisional diagnosis which is clinched only by X-ray examination.

Total pneumothorax without displacement may be diagnosed on careful physical examination, especially if one happens to find the metallic sound-phenomena, which are not always present. As a rule, but not always, the percussion note is tympanitic; it may be quite normal (No. 40).

In pneumothorax with moderate displacement the diagnosis is not so hard, as this condition also implies a distension of the affected side. But even here the diagnosis is by no means easy. And the longer the time between the onset of the illness and the examination, the more unusual the localisation of the initial pains, and the less pronounced the auscultatory phenomena, the more difficult is the diagnosis.

As to the first of these points, the dyspnoea, the pains and the tachycardia subside i a few days, as already mentioned, whereafter the only complaint of the patient is

often merely a slight shortness of breath on movements. As to the second point, under the symptomatology it has already been mentioned that not infrequently the initial pains may be so deceptive that neither the patient nor the physician will think of a lesion in the chest.

In two cases (No. 22 and 35) where the pains were localised to the shoulder on the affected side, the lesion was at first regarded as *rheumatism.* In one case (No. 6) that was ushered in with pains in the back, the patient thought she had *lumbago,* so that instead of consulting a physician to begin with, she went to a masseuse. In one case, No. 26 *a,* which has probably been an attack of tension pneumothorax, the pains were so violent and localised to the upper right part of the abdomen, that the lesion was taken to be a *perforated gastric ulcer,* and, consequently, laparotomy was performed.

Still, in 22 of the cases here presented the diagnosis was made by the family physician who sent the patient to the hospital — in some of the cases, though, only after X-ray examination.

12 cases have been regarded merely as acute chest-troubles of obscure nature — in two of these, though, with the possibility of acute cardiac insufficiency.

In 6 cases the diagnosis has at first been *pleurisy;* in fact, in two of these (No. 1 and 21), as mentioned before, friction sounds were also heard.

1 case has been regarded as *bronchopneumonia;* in 3 cases the lesion was at first taken to be *pneumonia.*

Finally, there are 2 cases in which the pneumothorax was discovered accidentally while the patients were under treatment for some other lesion (No. 32 and 35). In the last case, the previous attacks had been regarded as rheumatism.

Beside the two last-mentioned cases, there are 4 other cases (No. 8 *a,* 15, 22, 42 *a*), in which the diagnosis must have been particularly difficult, of course, as here the typical acute onset was absent.

Taken altogether, it may be said that the diagnosis is most easy in tension pneumothorax which presents a truly characteristic picture of the disease.

In chronic pneumothorax the diagnosis may be very difficult because the patients are so little inconvenienced by their lesion that, sometimes, they do not even realise themselves that something is the matter with them. In left-sided chronic pneumothorax with displacement of the heart one may then be inclined to take the abnormal position of the heart as situs inversus. In No. 32, with left-sided chronic pneumothorax, the left side of the chest was somewhat bulging in 1926, while on the after-examination, in 1929, it was decidedly flattened — though the heart was still in the right side — what may also be rather confusing.

Particular difficulties are encountered in the diagnosis of hæmopneumothorax. The collapse and anæmia of such patients are suggestive of other lesions. When located on the right side, hæmopneumothorax is also frequently accompanied by marked tenderness and rigidity of the upper part of the abdomen, which may lead to the assumption of a perforated gastric ulcer. When in these cases the examination has established the presence of pneumothorax with an accumulation of fluid in the pleural cavity, it takes of course an exploratory puncture to settle the nature of this fluid — whether blood or exudate.

In relapsing or in alternating pneumothorax the diagnosis is not so hard to make as in the first attack, and *often the patient makes the diagnosis himself* as soon as the same old symptoms appear.

In facing a case of bilateral spontaneous pneumothorax, which is indeed a very rare condition, it will naturally be a great help that one is acquainted with the features of this lesion and thinks of this possibility at once.

In cases of spontaneous pneumothorax with subcutaneous emphysema it will sometimes be very difficult to make out the presence of pneumothorax. In such a case as No. 48, in which the emphysema of the mediastinum and of the

subcutaneous tissue of the neck and chest was at first the predominant feature while the pneumothorax did not appear till later, the diagnosis of the latter condition is very hard to make, and it can be established only by puncture or by X-ray examination.

When one is to decide whether a case of spontaneous pneumothorax may be considered a clear-cut instance of the disease here discussed, Roentgenography is an indispensable requisite, and one cannot do with less than two X-ray examinations.

The first picture, taken as early as possible after the onset of illness, gives information as to the amount of air in the pleural cavity, and it allows of calculating the probable duration of the lesion. But, naturally, it gives no information as to the anatomical details in the affected lung as long as this is collapsed. Only a subsequent X-ray examination which should always be made after the affected lung has fully expanded, may give satisfactory information as to the possible presence of clinically nondemonstrable tuberculous processes in this lung. The last X-ray examination is therefore the most important with regard to the classification of the case and its prognosis.

In view of the diagnostic difficulties it is not unreasonable to assume that several cases of this disease are not diagnosed at all. Most likely, the lesion is not so very scarce, and several cases are probably recorded under some other diagnosis, or under the heading: Diagnosis uncertain.

PROGNOSIS

In no form of pneumothorax is the outlook so favourable as in spontaneous pneumothorax in the apparently healthy. This applies to the physical restoration of the patient as well as to his life. The good prognosis is therefore one of the most characteristic features of the disease.

None of the 51 patients here reported has died from pneumothorax.

Amongst the ca. 200 typical cases of spontaneous pneumothorax reported in the literature only 6 have terminated in death. These six are the following cases:

1) *Desmaroux* (1884): Male, aged 25. Sudden onset of *tension pneumothorax* with collapse and extreme dyspnoea. Death on the 3' day of illness. The author says himself that the life of the patient might perhaps have been saved if puncture had been performed in time. No autopsy.
2) *Pitt* (1900): *Hæmopneumothorax* (see p. 20.).
3) *Rolleston* (1900): *Hæmopneumothorax* (see p. 90.).
4) *Hayashi* (1915): *Bilateral pneumothorax* (see p. 21.).
5) *Fischer* (1922): *Hæmopneumothorax* (see p. 21.).
6) *Kiær* (1923): *Hæmopneumothorax* (see p. 91.).

Therefore, the death-rate is certainly very small, provided that the proper treatment is given. For several of the other cases have implied a very serious danger of death — cases in which the lives of the patients were saved only through a well-calculated treatment.

The forms of the disease involving a danger to life are:

1. *Tension pneumothorax.*
2. *Hæmopneumothorax.*

3. *Bilateral pneumothorax.*
4. *Pneumothorax associated with severe subcutaneous and mediastinal emphysema.*

In *tension pneumothorax* one must always keep in mind that death may come suddenly. When, nevertheless, there is reported but one case where this has happened, it is owing to the fact that in this form of pneumothorax the disease is relatively easy to recognise, and that puncture is performed as soon as the diagnosis is made. That these patients are nearly always young persons without any heart trouble, is further contributory to the good prognosis.

A patient with *bilateral pneumothorax* will be suffocated if not relieved at once. In the case reported by Hayashi, the patient died, whereas Elte succeeded in saving the life of such a patient.

A combination of suffocating mediastinal emphysema and pneumothorax is likewise involving a great immediate danger to life. Yet, in the case here reported — No. 48 — the attending physicians succeeded in curing the patient.

Apart from these 4 relatively rare forms of pneumothorax, the lesion does not imply any danger of death, except in so far as every case of pneumothorax involves an indirect danger in this respect, that the patient will succumb readily if he has an attack of pneumonia in the other lung.

As to the full return of *physical ability* the outlook is also very favourable.

In my material of 51 patients with at least 61 attacks of pneumothorax, there is only one patient, No. 32, in whom the pneumothorax persisted for ca. 20 years; and even in this case the lung did finally begin to expand. All the others recovered from their pneumothorax.

As to the duration of the illness, this has already been mentioned under each form of pneumothorax. In 4 of my patients, the disease has taken a chronic course, result-

ing ultimately in spontaneous recovery (3 cases) or marked improvement (1 case) even though the lesion has lasted a good many years.

We then arrive at the very important question:

How are these patients getting on later in life?

As to the later condition of these patients, the literature gives but very few data. These are:

Mc.Dowell's	patient	was	well	3	years	later.
Liisberg's	„	„	„	7	„	„
West's(4)	„	„	„	10	„	„
Rosenbach's	„	„	„	10	„	„
Fussell & Riesmann's	„	„	„	7	„	„
Mc.Kie's	„	„	„	2	„	„
Hamman's 2'	„	„	„	4	„	„
Huet's	„	„	„	6	„	„
Cahn's 2'	„	„	„	6	„	„
Schüle's	„	„	„	3	„	„
Joerdens'	„	„	„	4	„	„
Le Wald's(1)	„	„	„	2	„	„
Dahlstedt & Haeger's	„	„	„	3	„	„
Enneking's 1'	„	„	„	18	„	„
Enneking's 2'	„	„	„	16	„	„
Enneking's 3'	„	„	„	11	„	„
Enneking's 4'	„	„	„	11	„	„

In addition, *Zinn & Siebert* have reported 8 cases in which the patient kept on being well, but half of them had been under observation only a very short time.

In 1914, Lindhagen published a case in which the patient showed pulmonary tuberculosis 7 years after his attack of spontaneous pneumothorax. But as in this case the X-ray examination had revealed, even at the time of the

spontaneous pneumothorax, a considerable diffuse density of the central part of the lung tissue, which was most likely of tuberculous nature. I think, as pointed out before, that such a case should not be included under the heading: spontaneous pneumothorax in the apparently healthy.

According to a report of Tillisch, the aforementioned patient of Bull, with relapsing spontaneous pneumothorax, has later died of pulmonary tuberculosis. As to this case, one may say that inasmuch as the patient has not been examined with X-rays because this method of examination did not exist at that time, it is possible that the patient may have had a latent tuberculosis even at the time when the spontaneous pneumothorax set in — just like in Lindhagen's case.

Faschingbauer has reported a case of bilateral partial pneumothorax — presumably due to true bullous emphysema of the lungs — in which the patient had subsequently pulmonary tuberculosis. But this case falls outside the clinical entity here discussed.

Hamman, on the other hand has described a typical case of alternating pneumothorax (Pt. No. 1), in which an attack of pneumothorax on the right side was followed by 2 attacks on the left side. During the first and the last attacks the patient gave negative tuberculin reactions. 2½ years after the last attack, the patient had pulmonary tuberculosis.

In addition, Mosheim has reported a case (No. I) in which the patient showed pulmonary tuberculosis 4 years after an attack of spontaneous pneumothorax. As the author says, one may reckon with the possibility that here the tuberculosis may be due to a subsequent infection. But pleural effusion was also present in this case so that it is not any clear-cut instance of the lesion under discussion here.

As far as I have been able to find out, these cases are the only ones in the literature wherein a patient with this form of spontaneous pneumothorax has acquired pulmo-

nary tuberculosis later on. Amongst these cases, the one reported by Hamman is really the only one that meets the definition and delimitation of this clinical entity as here laid down. And such an isolated instance does really mean but very little — it seems reasonable to take it as an example of accidental coincidence.

Naturally, a tabulation of solitary cases gathered from the literature will never give any true picture of the prognosis of a disease, as the great majority of reported cases are peculiar cases, differing from the usual clinical picture in some way or other. They are therefore selected cases which are not suitable for statistical estimates, and they do not represent the *marche naturelle* of the disease.

The burning question is whether or not these patients acquire pulmonary tuberculosis later on.

It has been, therefore, the principal aim of the work here presented to give a contribution to the settling of this question. So I have collected as large a material as possible, and afterwards I have looked up the patients in order to obtain information as to the later condition, and I have made after-examination — including Roentgenography — of them.

The result of these after-examinations is as follows:

Amongst my 51 patients, 2 (No. 2 and 34) could not be found (moved away, address unknown).

As to the remaining 49 patients, complete information is obtained about their health after the pneumothorax attack.

3 of the patients have died from various cause.

One of these, No. 12, had 2 attacks of pneumothorax, 8 months apart. He was then well for 3 years till he had an attack of lobar pneumonia, of which he died.

One patient, No. 21, had spontaneous pneumothorax in the last part of August, 1923. 5 months after his discharge from the hospital, he acquired pneumonia which was subsequently complicated with empyema and pyopneumothorax; and the patient died from this disease. On autopsy, no tuberculosis could be made out in the lungs.

The third one of the patients who have died (No. 24) is mentioned in detail below.

Amongst the remaining 46 patients, two — No. 14 and No. 50 — are now residing abroad, so that I have had to be content with their written statements as to their present condition; but, besides, they have both been examined several times by other physicians, who have declared them to be well.

This leaves 44 patients who have been after-examined — some of them even several times — and X-ray plates of the lungs have been taken in every instance.

So the result is that in 49 cases with complete data there is only one patient who has acquired pulmonary tuberculosis after the attack of spontaneous pneumothorax.

This patient — No. 24 — had in March 1924 an altogether typical attack of spontaneous pneumothorax on the right side, probably the fourth attack in this patient. After this, the patient — female, aged 25 — was well for 3 years, till she had an acutely developing, severe, degree of progressive pulmonary tuberculosis with cavity formation of the left lung (she belonged to a family of consumptives). No abnormality could be demonstrated in the right lung. As the patient kept on getting worse, artificial pneumothorax was performed on the left side, with good effect. After the artificial pneumothorax had been maintained a little over 7 months, the patient had, in connection with an acute febrile disease, again an acute attack of spontaneous pneumothorax on the right side, and she died. Autopsy: On the left side of the chest, the artificial pneumothorax occupies about one half of the pleural cavity. The upper lobe of the left lung consists almost entirely in a thick-walled cavity filled with purulent material. The lower lobe of the left lung shows many tuberculous foci. The right pleural cavity is the site of spontaneous pneumothorax, the right lung being compressed to half its natural size. There is no fluid in the pleural cavity. The site of the pleural rupture can not be made out. The middle lobe

of the right lung shows many tuberculous foci. The right lower lobe shows emphysema — and at the base of this lung, resting upon the diaphragm and protruding laterally out into the air space, is a very thin-walled vesicle (about the size of a goose-egg) consisting of greatly distended lung tissue.

It seems to me that the most natural interpretation of this history will be about as follows:

Through a number of years, the patient has had a valve vesicle at the base of the right lung that has given rise to the first 4 attacks of spontaneous pneumothorax. This view is based on the autopsy findings: emphysema of the lower lobe of the right lung, with a very thin-walled vesicle as large as a goose-egg. Although the lung is compressed to about half its natural size, this vesicle is still distended; and this suggests very strongly that it is a valve vesicle. It is then quite natural to think that it must have been ruptures of this enormous vesicle that have given rise to the first 4 attacks of spontaneous pneumothorax.

3 years after the fourth attack of spontaneous pneumothorax, the patient has an acutely developing phthisis of the left lung with cavity formation.

In view of the facts, 1) that the spontaneous pneumothorax and the subsequent condition of pulmonary tuberculosis in this patient are localised each to its separate side, 2) that she is perfectly well — clinically — during the 3 years intervening between the two lesions, and 3) that she comes from a home where tuberculosis is prevalent, the evidence goes to show that these two lesions in this patient are not parts in the development of the same morbid condition, and I think it warrantable to consider the two lesions independent of each other.

The presence of numerous tuberculous foci in the middle lobe of the right lung, as revealed on autopsy, is explained most naturally as disseminations from the far-advanced tuberculous process in the left lung. Whether the spontaneous pneumothorax may have been due to the

rupture of one of these small tuberculous foci in the middle right lobe, or whether it has been due to a rupture of an emphysematous vesicle in the right lower lobe, this question is not cleared up by the autopsy, as no rupture of the pleura could be demonstrated.

But apart from the fact that this interpretation of the case record appears to me to be the only probable explanation of the case, this single instance of subsequent pulmonary tuberculosis in a material of 49 after-examined pneumothorax patients does really mean but very little in comparison with all the 48 cases where no tuberculosis could be made out. Nor was it to be expected that all patients with spontaneous pneumothorax would later turn out, so to speak, to be immune to pulmonary tuberculosis.

The result of these after-examinations are presented schematically below.

Most of these patients have been perfectly well since their recovery from pneumothorax. A few of them have been troubled with various diseases that have nothing to do with pneumothorax — e. g., hypertrophy of the prostate, phlebitis, etc. Such lesions being irrelevant to the question under analysis, they have not been taken into account in estimating the condition of the patient on the after-examination. If otherwise he has been well, he is put down as well.

All the patients characterised as well have been feeling well — without any symptoms of tuberculosis — ever since they had their attacks of pneumothorax, and the after-examinations as well as the X-ray plates have shown that they are in good health objectively too. In some of these patients the after-examination has shown a slight extension of the lung borders, indicating a very mild degree of emphysema that has been acquired as the years went by, without giving any discomfort; these are also entered

as well. The number of years recorded in the schema signifies in each case the time that has passed from the pneumothorax of the patient was recognised as such till the (last) after-examination. The patients are entered according to the date when their pneumothorax was diagnosed.

SURVEY OF THE AFTER-EXAMINATIONS

No. 1: Well during the past 18¾ years.
No. 2: Not found.
No. 3: Well during the past 16¼ years.
No. 4: „ „ „ „ 15¼ „
No. 5: „ „ „ „ 14¾ „
No. 6: „ „ „ „ 14½ „
No. 7: „ „ „ „ 14 „
No. 8: „ „ „ „ 13 „
No. 9: „ „ „ „ 10¾ „
No. 10: „ „ „ „ 9½ „
No. 11: „ „ „ „ 9 „
No. 12: Well during 3½ years. Then he died of lobar pneumonia.
No. 13: Well during the past 8¾ years.
No. 14: „ „ „ „ 8 „
No. 15: „ „ „ „ 7½ „
No. 16: „ „ „ „ 7½ „
No. 17: „ „ „ „ 6 „
No. 18: „ „ „ „ 6 „
No. 19: „ „ „ „ 5¼ „
No. 20: „ „ „ „ 5¼ „
No. 21: Well during 5 months. Then he died of pneumonia with pyopneumothorax.
No. 22: Well during the past 4¾ years.
No. 23: „ „ „ „ 4¼ „
No. 24: Well during 3 years. Then tuberculosis of the other lung.

No. 25: Well during the past 4¼ years.
No. 26: „ „ „ „ 4¼ „
No. 27: „ „ „ „ 3¾ „
No. 28: „ „ „ „ 3 „
No. 29: „ „ „ „ 3 „
No. 30: „ „ „ „ 3 „
No. 31: „ „ „ „ 5½ „
No. 32: „ „ „ „ 5½ „
No. 33: „ „ „ „ 5½ „
No. 34: Well during 1 year. Then he moved away — address unknown.
No. 35: Well during the past 4½ years.
No. 36: „ „ „ „ 4½ „
No. 37: „ „ „ „ 4½ „
No. 38: „ „ „ „ 4½ „
No. 39: „ „ „ „ 4¼ „
No. 40: „ „ „ „ 4¼ „
No. 41: „ „ „ „ 4¼ „
No. 42: „ „ „ „ 4¼ „
No. 43: „ „ „ „ 3¾ „
No. 44: „ „ „ „ 3¾ „
No. 45: „ „ „ „ 3½ „
No. 46: „ „ „ „ 3½ „
No. 47: „ „ „ „ 3½ „
No. 48: „ „ „ „ 3¼ „
No. 49: „ „ „ „ 3¼ „
No. 50: „ „ „ „ 2¼ „
No. 51: „ „ „ „ 2 „

Thus, as evident from this survey, *all these patients — except the last two — have an observation period of at least 3 years, and the examination of the oldest cases gives the same findings as does the examination of the most recent ones.*

The result of these after-examinations, I think, may therefore be said to be reliable and in accordance with the

general run of this disease, for my material is so large that this result can not be altogether accidental.

This is significant, not only as to the prognosis, but also with regard to the whole view of this disease as a lesion of benign character. For this much is really implied in the result of these after-examinations.

Of course, it is not impossible that a patient with spontaneous pneumothorax — as this lesion is here defined — may later have pulmonary tuberculosis, as they are no more immune against tuberculosis than are other persons. *But my after-examinations have shown that the probability of this event is so slight that it may be left out of account in practice.*

THERAPY

The treatment that has been employed in the cases here described has naturally been varying to some extent, partly because it had to be adjusted to the degree — mild or severe — of the lesion, and partly because, in some cases, it has been influenced by different points of view as to the etiology and prognosis of the disease. Thus, in some cases the treatment has been aimed to maintain the condition of pneumothorax for some time by means of insufflation, whereas in other cases the therapeutic efforts have been directed towards a shortening of the course of the disease by aspirating some of the pneumothorax air.

In the great majority of the cases, however, the treatment has merely consisted in keeping the patient in bed a couple of weeks or more, according to the severity of the case, followed by a suitable period of convalescence.

On account of tension pneumothorax it has been necessary to puncture the pleural cavity shortly after the onset of the illness in 4 of the patients (No. 11, 16, 26 *a*, and 36), besides in the patient with subcutaneous emphysema and pneumothorax (No. 48).

In 4 cases, where there were no urgent indications for a puncture, this measure was yet employed in the beginning of the disease to give the patients some relief (No. 26 *b*, 31, 42 *b*, and 42 *c*).

In No. 23, who had hæmopneumothorax, aspiration was performed on the 18' day of illness, with evacuation of some of the blood.

In 4 cases (No. 1, 42 *a*, 50, and 51) aspiration of the

pneumothorax air was done subsequently in order to shorten the course of the illness.

From the consideration that they were suspicious of harbouring a latent, non-demonstrable, pulmonary tuberculosis, 5 of the patients have been treated with air insufflation in order to maintain their condition of pneumothorax, so that the supposedly underlying affection might heal (No. 8, 14, 20, 23, 25). In 4 of these cases, insufflation was given but a few times; and only in one case (No. 8) was this treatment carried out rationally, through a period of 2½ years. The last-mentioned patient was at first treated in a tuberculosis sanatorium.

From the same consideration, 10 of the other patients have been under after-treatment in tuberculosis sanatorium (No. 2, 15, 20, 22, 29, 30, 35, 38, 40, 45).

In turning to the treatment of the various forms of this lesion, I may briefly mention the question of *prophylaxis*.

The rupture itself cannot be prevented, but if the diagnosis is made early, and if the patient is kept in bed from the beginning of the illness, it will be possible in some cases to prevent that the lesion progresses to a severe degree. For, as pointed out above, in some cases the very severe degrees of the lesion do not develop till the patients have been going about for a couple of days with their pneumothorax. So the early diagnosis is important, as it happens but seldom that the amount of pneumothorax air will further increase after the diagnosis is made — that is, when the patient is kept in bed.

As mentioned before, relapse of the lesion occurs in about 20 per cent of the patients, and in more than half of these cases, the relapse comes within the first year. If the relapses are due to new ruptures of the same valve vesicle — what one cannot know for sure, however — it may be

possible to prevent such relapses by having the patients avoid any form of physical strain throughout the first year, at the least.

As it is very doubtful, however, whether any benefit could actually be derived from such precautions, it will be sufficient to tell the patients that relapses may occur, without laying down any restrictions for their work or advising any change of occupation. Strenuous sport, on the other hand, and in particular swimming, should be forbidden. Of course, if one is dealing with one of the very rare cases, in which relapses set in again and again, one must urgently advise the patient to be careful of himself.

PARTIAL PNEUMOTHORAX

The treatment consists in having the patient stay in bed 2 weeks, so that one may reckon with the perforation having healed when he gets up. If there are severe pains in the beginning of the disease, aspirin or pantopon may be prescribed. In addition, one must deal with any condition that may give rise to an increase of the intrathoracic pressure, and thus bring about an aggravation of the pneumothorax. The point is therefore to relieve any coughing and avoid any constipation. The patient should not return to any physical work till at least 3 weeks after the onset of the illness.

COAT-FORMED PNEUMOTHORAX

Here the treatment is quite the same as in the partial pneumothorax.

TOTAL PNEUMOTHORAX WITHOUT — OR WITH VERY SLIGHT — DISPLACEMENT

In the beginning of the illness, severe pains may sometimes call for small doses of a narcotic. Otherwise, confinement to bed for a couple of weeks will be sufficient.

If in the next 2 weeks the patient can keep from working, most of the pneumothorax air will be absorbed, as a rule. But if it is urgent for the patient to return to his work as soon as possible, or if there is no sign of the pneumothorax air being absorbed, one may perform aspiration — though not until at least 2 weeks after the onset of the illness. For at that time one may expect the lung to be in beginning expansion, with an intrapleural pressure around 0, or below, although the possible negative pressure will not be as low as the normal negative pressure. In such cases, the pneumothorax is now closed off, and the perforation may be taken to have been filled in organically.

Most authors are of the opinion that aspiration should not be done too early. Only one author, von Teubern, has employed aspiration in the first days after the onset. But as one can not reckon with the perforation having closed organically till about 2 weeks after the onset of the illness — cf. No. 42 *b* — an earlier aspiration may readily bring about that the perforation opens again.

Aspiration must be performed slowly, cautiously and under manometer control — no matter whether one employes a Dieulafoy syringe or a pneumothorax apparatus. Israel-Rosenthal recommends a cautious "fractional" withdrawal of ca. 500 cc. of air each time, such as it was done in the case of No. 1, who was one of Professor Israel-Rosenthal's patients.

Tallquist emphasizes, too, that the aspiration must be carried out slowly and carefully — if possible, in several seances — on account of the risk of shock and pulmonary oedema.

After the aspiration there may be slight cyanosis, and there may also be fine râles in the expanded lung as a sign of a mild degree of pulmonary oedema, but this subsides rapidly.

TOTAL PNEUMOTHORAX WITH MODERATE DISPLACEMENT

Here the treatment must follow the same lines as in the preceding form of total pneumothorax. In these cases it takes even longer time for the complete absorption of the air, on an average from 6 weeks to 2—3 months. Consequently, one may here consider more frequently the expediency of an aspiration, but if the patient is not troubled all too much with dyspnoea on moderate movements, there will naturally be no urgent reason to shorten the absorption time. Instances of curtailment of the disease by means of aspiration are found in cases No. 42 *a*, 50 and 51.

TENSION PNEUMOTHORAX

In tension pneumothorax, the necessity of prompt and energetic treatment is far more urgent.

There has been published only one case in which an apparently healthy person has died from tension pneumothorax. But this low mortality is undoubtedly due to the fact that as a rule, in this condition, the diagnosis is made at once, so that puncture may be performed in time.

As soon as there is the least sign of tension pneumothorax — whether it is cyanosis, profuse sweating, collapse, marked dyspnoea with a respiratory rate above 30, marked bulging of the affected side, or marked displacement — this constitutes an indication for puncture.

The pulse rate is usually above 100, but it may also be normal, and one must not rely too much on the slow pulse and take it to mean that all is safe. Even though the pulse is slow, strong and regular, a case of tension pneumothorax may yet end with heart failure at any time (Møllgaard).

The puncture is made with a thin cannula. This may be connected with a rubber tubing leading, under water, to a container where the evacuated air may be collected and measured.

The puncture will always give the patient a great relief: the anxiety and the pain subside, the cyanosis fades away, the dyspnoea falls off, the heart moves back towards its normal site, and the patient feels an altogether different man (cf. fig. 21 and 22).

By this, as a rule, these patients will usually be on the road to recovery, and they will be able to get along with this one puncture. In some cases, however, it will be necessary to repeat the puncture, or to leave the cannula in the chest wall, and therefore it will be best to have such patients admitted to the hospital, where one can be sure they will be attended to immediately if new tension symptoms were to develop.

In very severe cases of tension pneumothorax it may be necessary to employ the method given by P. Bogason for permanent drainage of the air with automatic pressure regulation (water-trap cannula). With this method, Bogason has obtained some very beautiful results in treatment of spontaneous pneumothorax in pulmonary tuberculosis.

If the patient is in an exhausted state, some stimulant is given. It is very important to make the patients lie quietly and to relieve them of their pains, so that they are not coughing or breathing with painful and jerky expiration through a glottis that is partly closed, which will only pump more and more air through the valve opening. Patients who are restless, anxious or markedly distressed, should, therefore, always have morphine or pantopon.

As soon as the first, critical, days are over, the disease will usually come down into a state of quietness, and the patient may be able to get up after keeping his bed for 2—3 weeks. The treatment will then be the same as in other forms of total pneumothorax; here too, one will have to consider the expediency of shortening the course of the illness by subsequent aspiration, for as a rule it takes about 2—3 months before the air is absorbed.

CHRONIC PNEUMOTHORAX

In some of the cases where the pneumothorax has persisted more than a year, one must assume that the perforation stays patent (cf. No. 32). In other cases it is more reasonable to assume that the perforation is closed at times and open at other times — like a valve (cf. p. 57).

The characteristic of this form of pneumothorax is the fact that it troubles the patients so little that, sometimes, the lesion is discovered only by chance.

Here the possibilities of recovery are as follows:

1) Spontaneous recovery.
2) Treatment with repeated, cautious, aspirations.
3) Treatment with artificial production of aseptic pleurisy by means of intrapleural injection of glucose solution.

As shown in the cases of this material, spontaneous recovery takes place in a good deal of these patients after they have been going about with their pneumothorax for a couple of years. This happened in 3 out of my 4 cases (No. 4, 6 and 35). In the fourth case — No. 32 — where the pneumothorax has persisted ca. 20 years, the lung has finally now, within the last year or so, expanded in part, so that the lesion is now a partial pneumothorax.

This fact is naturally of great significance. One will, therefore, be disinclined to employ any radical treatment

in these cases, and the more so, as the patients are inconvenienced but very little by their lesion. Yet, indeed, the fact that one lung is put out of function involves a certain danger, as illustrated in Brunner's first case (cf. p. 21.) where the patient contracted influenza-pneumonia in the healthy lung, and died. Moreover, chronic pneumothorax may bring about a deformity of the thorax or formation of adhesions, as is evident from the record of No. 6.

If, then, the chronic pneumothorax keeps dragging on, one should try to further the process of recovery by means of repeated cautious aspirations.

Even though it would offhand seem reasonable to think that aspiration could be of no avail as long as there was still a perforation — even if very small — in the pleura, this method has yet given some good results in a few cases. Schreiber and Emil Als have both succeeded in bringing a chronic pneumothorax to recovery through this method. So it is always worth while to make a try with this method, especially as it is so easy to carry out.

If one does not deem it prudent to wait any longer, and if the aspiration treatment does not lead to any improvement, one may try to produce an artificial pleurisy by injection of glucose solution, into the pneumothorax cavity.

Attempts have also been made to bring about a healing of the perforation by intrapleural injection of venous blood. This was done in Brunner's second case (cf. p. 22.), but of no avail. Recently the method has been employed by Watson & Robertson in a case of persistently relapsing spontaneous pneumothorax. In this case it seems to have been of some use, but the authors think that nothing definite may be said about the value of the method merely on the basis of this one case.

In the other cases of chronic pneumothorax where the injection treatment has been tried, glucose solutions have been employed for injection fluid.

The method was first suggested by Lucius Spengler for treatment of protracted pneumothorax without any tenden-

cy to spontaneous recovery. One begins with injecting 30 c.c. of a hypertonic glucose solution (30 %), of which the osmotic pressure is about 5 times that of the blood, and this gives rise to a congestion of the pleura with effusion, resulting in a small exudate.

Spengler[2]) reports a case of pneumothorax in which, he claims, this treatment proved successful. The exudate was only slight, merely filling the complemental space, and it took 4 months before the pneumothorax was all gone. So it is possible, indeed, that the lesion might have subsided spontaneously. In his latest paper, 1923, Spengler states that, if necessary, one may use a more concentrated solution of glucose (40—50 %), or inject the 30 % solution in larger amounts, up to 100 c.c.

Kulcke has employed this treatment in the case of a 9-years-old girl who had had pneumothorax for 3 months. The injections were given 7 days apart, in increasing doses: 20, 35 and 50 cc. of a 30 % glucose solution. The injections were followed by a rise of temperature, up to 38.2. The injections were painful, and produced dyspnoea. So the treatment was limited to these 3 injections. 6 weeks later, the pneumothorax had disappeared.

Brunner has also made a try with this method — in the aforementioned case (p. 21.) — but the treatment was not carried through.

Fogelberg & Wallgren have treated two cases of pneumothorax with glucose injections.

Upon the whole, it may be said that the results of the glucose treatment are not particularly convincing, even though a small exudate may actually be produced with this method. The treatment is probably safe, and yet it may give rise to pain, dyspnoea and a transitory rise of the temperature. In fact, Fogelberg & Wallgren recommend to give morphine before the glucose injection.

In view of the fact that the perforation in most instances are located in the apex, it will be expedient to elevate the foot-end of the patient's bed if this does not

give him dyspnoea, so that the glucose solution may exert its effect in the apical part of the pleural cavity — the most frequent location of scar-tissue vesicles.

However, one may hardly expect to obtain recovery in all cases of chronic pneumothorax. When this lesion has been present since childhood — as in No. 32, in whom it has persisted for about 20 years — it is not very likely that the full functional capacity may ever be restored, as the lung tissue and the blood vessels are probably not developed in the compressed lung to the same extent as on the healthy side.

HÆMOPNEUMOTHORAX

In mild cases of hæmopneumothorax, where the hæmorrhage is very slight, this bleeding may be disregarded in the treatment. In No. 36, the clinical appearance of the disease was that of a tension pneumothorax, and thus it had to be treated as such a lesion without regard to the small bleeding that was present too (see fig. 21 and 22).

If, on the other hand, one is dealing with a patient in whom the hæmorrhage is severe — from about one up to several liters of blood — one is facing a very serious lesion, the diagnosis and treatment of which may be very difficult. These patients are always showing symptoms of collapse from hæmorrhage; and it is the symptoms of collapse, more than the pneumothorax symptoms, that are the predominating features in the clinical picture of these patients. The acute collapse will usually make its appearance several hours after the lung rupture has taken place.

The seriousness of this disease is evident from the fact that in the 10 aforementioned cases the bleeding has stopped spontaneously only in 6, whereas 4 of these patients died from the hæmorrhage. All these cases were treated expectatively.

Is goes without the saying that everything must be done to make it as comfortable and quiet for these patients as possible, that any unnecessary examinations are avoided once the diagnosis is made, and that morphine must be given if the patient is restless.

Intravenous injection of salt solution and blood transfusion must be said to be *contraindicated,* as it is to be feared that an increase of the blood pressure may bring about an aggravation of the hæmorrhage. For the same reason, cardiac stimulants are also to be avoided.

If, on continuous observation, the symptoms show that the patient keeps on bleeding, so that it is to be feared he will die, one might consider the advisability of thoracotomy in order to try to locate and ligate the bleeding vessel.

It takes great experience in surgery to decide whether such a measure will give the patient a chance of surviving, and whether such an extremely exhausted patient will be able to go through such a great operation. As already mentioned, the operation has not been performed in any of these cases, but, judging from the surgical treatment of injuries to the lungs, there can be no doubt that it is a difficult operation. In view of the fact that in the aforementioned cases of Rolleston and of Rudolf Kiær it was not possible on autopsy to find the site of the perforation, one should also keep in mind that it may be impossible to find the site of the hæmorrhage if an operation is resorted to.

Of course, such an operation can be performed only in a hospital. Still, the question, whether it is not too risky to move these patients, will often be solved in practice in this way, that the diagnosis is not made till the patient is admitted to the hospital.

If one succeeds in making the diagnosis in the patient's home, one must realise that the transport of such a patient involves a very serious risk.

Lejars points out most emphatically that in injuries to the lungs nothing is so fateful as a troublesome transport

of these patients, who above all require absolute rest. The same point is emphasized by Wieting.

Although the experiences concerning hæmopneumothorax are but few, they still seem to show that the chances are most in favour of a spontaneous cessation of the bleeding. In the one case included in my material, No. 23, the patient was not moved till the 3' day of illness, and his transport was the easiest possible, as he lived just opposite the hospital. It may be that this transport would have been fatal if he had been moved in a hurry-scurry the same evening he collapsed from the loss of blood.

On the other hand, it is obvious that if the patient is steadily getting worse, so that it will be necessary after all to risk a thoracotomy in order to try to save his life, it will not do to postpone the moving of the patient, till he is on the point of dying, if he is to go through the transport and the operation both.

If the patient survives the first, critical, days, the treatment will usually not give any particular difficulties. If the absorption of the blood is very protracted, it may be useful later to evacuate this blood — as it was done in No. 23, on the 18' day of illness. Naturally, the anæmia resulting from the hæmorrhage is to be treated in the usual way.

Further, it will be the proper thing to tell these patients explicitly to avoid all exertion, at least the first year or so, on account of the risk of a relapse.

RECURRENT PNEUMOTHORAX

The relapses are treated after the usual principles, but the patients should be advised to avoid any severe exertion during the first year after the relapse, as one has to reckon with the possibility of more relapses.

In those rare instances where — as in Sale's case — the relapses keep on coming in rapid succession, the treat-

ment must naturally aim to prevent these relapses. And this would be attained if one could produce an artificial pleurisy resulting in adhesions that kept the lung from collapsing. In such cases one will therefore make a try with the method of glucose injections, as mentioned above under the chronic pneumothorax.

This treatment is the rational therapy in such cases, and it may be considered quite safe, so that it ought to be tried out during one of the relapses in these patients.

ALTERNATING PNEUMOTHORAX

Cases of alternating pneumothorax, in which there is but one attack on either side, will usually not call for any particular measures. If, on the other hand, one is dealing with cases so severe as the two reported by Watson & Robertson — case I and case III — where the pneumothorax makes its appareance again and again, now on one side, now on the other, one may also here make a try with glucose injections.

Patients with alternating pneumothorax should always be brought to the hospital, on account of the dangerous possibility that the lesion may make its appearance on both sides at the same time.

BILATERAL PNEUMOTHORAX

If a patient has a severe degree of bilateral pneumothorax, as in the aforementioned case of Elte, every minute is precious. Permanent aspiration should be instituted as quickly as possible, by means of a water suction pump, or an electrical suction pump. As recommended by Elte, one begins with puncture and aspiration on that side where the pneumothorax has been present the longest, and if

this is not sufficient, the operation is performed on the other side, too, so as to make it a bilateral permanent aspiration (see p. 102.).

If there are no accessories at one's disposal, one must make the experiment to attach a piece of rubber tubing to the cannula and carry out the aspiration with one's own lungs. Of course, a large syringe or a rubber bulb may also be of use in an emergency.

Watson & Robertson have treated one case in which a total of 14 pneumothorax attacks made their appearance within a couple of years — now on the right side, now on the left. At one time this patient had a considerable pneumothorax on the right side and, coincident with this, a partial pneumothorax on the left side over the collapsed apex. In this case the aspiration was limited to the right side where the pneumothorax was most pronounced.

PNEUMOTHORAX ALTERNATING WITH PNEUMOPERICARDIUM

The treatment of these two lesions does not call for any other measures than those already mentioned. The patient must be kept in bed for 2—3 weeks, until the perforation has closed and the absorption of the air from the pleural cavity or the pericardium is progressing.

PNEUMOTHORAX WITH SUBCUTANEOUS EMPHYSEMA

Very mild cases, as No. 8 *a,* do not require any special treatment, neither for the emphysema nor for the pneumothorax.

In No. 48, we meet with a lesion of a much more serious character — a suffocating mediastinal emphysema + spontaneous pneumothorax. And the life of this patient

was saved solely because the family physician, Dr. M. Sögaard Andersen, made the diagnosis at once, and because the treatment employed in the Svendborg Hospital, by the physician in chief, Dr. med. V. Reinsholm, was calculated so well, meeting all the exigencies of the case.

In this unique case the treatment consisted in repeated punctures of the pleural cavity, inhalation of oxygen and stimulation — all of which is mentioned in detail in the case record. In desparate cases one may further resort to the treatment which W. Hunter recommended as early as 1762: incision and expression of the air by gentle stroking of the emphysematous region. In such a case it will be best to make a small vertical incision in the skin over the jugulum.

AFTER-TREATMENT OF THE PATIENTS

Spontaneous pneumothorax in apparently healthy persons does not call for any after-treatment, and consequently, there is no need of sending these persons to a tuberculosis sanatorium.

When the prognosis, as demonstrated through this material, is so favourable for these patients with regard to their health later in life, one ought to save them the time and expenses of a stay in some sanatorium and the fear of pulmonary tuberculosis.

The presupposition of this standpoint is, of course, that the clinical features of the case in question fall along the lines of the definition of the lesion as here laid down. If, what will happen but seldom, one may have some doubt about the case, a stay in the country will be sufficient after-treatment.

SUMMARY

In the literature are reported about 200 cases of spontaneous pneumothorax in apparently healthy persons, but the various authors are not in agreement as to the conception of this disease.

On the basis of a material comprising 51 patients with a total of 61 attacks of pneumothorax, a contribution is here given to the etiology, symptomatology, diagnosis, prognosis and treatment of this disease.

ETIOLOGY AND PATHOGENESIS

The literature comprises 6 cases in which this lesion has been elucidated anatomically on autopsy. In these cases the post-mortem examination showed that the cause of the pneumothorax has been a rupture of superficial air vesicles in the apices of lungs which were otherwise healthy. Hayashi and Fischer, who described 3 of these cases, have demonstrated that these solitary air vesicles were formed on a base of cicatricial changes in the apices of the lungs; and the microscopic examination showed that the base of these vesicles includes a valve-like structure, which causes that these vesicles are gradually being distended more and more, so that finally they rupture.

According to Fischer, this disease would then be due to the rupture of such a valve vesicle, formed on the basis of a small scar in the apex of the lung.

My own studies on 3 cases of valve vesicles, found incidentally on autopsy, show that there are two forms of valve vesicles, widely different in origin and structure, which, by rupturing, may both give rise to spontaneous pneumothorax.

Both forms arise because at the base of the vesicles there is a valve-like structure. In the first of my cases, this valve mechanism was formed entirely through local emphysematous changes; and the valve itself was demonstrated on microscopic examination. In my second case there were several valve vesicles, resulting from shrinkage of scar tissue after the healing of a small tuberculous process in the apex of the lung. The third case showed a solitary scar-tissue vesicle in the apex of the lung; and here, too, the microscopic examination revealed the presence of a flap valve.

Thus there are valve vesicles with and without scar tissue — what has also been observed by Hayashi.

On X-ray examination, it has been possible to demonstrate the valve vesicle on the X-ray plate in one case (No. 42 *c*) of my material. Here it presents itself as a vesicle as large as a hen's egg, at the base of the lower lobe of the right lung. In this unique case, the greater part of the wall of the vesicle is most likely of fibrous character, and thicker than the wall of the valve vesicles in general. This will in part explain how this vesicle is demonstrable on the X-ray plate. Further, the vesicle is sufficiently large to show as a freely protruding body in the pneumothorax cavity. The patient has had altogether 3 attacks of right-sided spontaneous pneumothorax.

So the result of these findings is:

The disease is due to a rupture of a valve vesicle on the surface of the lung. There are two forms of valve vesicles. One form, the scar-tissue vesicles, develops especially in the apices of the lungs, next to a scar that may originate from healed tuberculous processes. These scar-tissue vesicles are often multiple. The other form, the emphysematous valve vesicles, is formed alone through local emphysematous changes without scar tissue. These may be found as large solitary vesicles on the margins of the lungs. Probably the scar-tissue vesicles constitute the more frequent form of valve vesicles.

One of my patients was later examined post mortem. The autopsy revealed no macroscopic signs of tuberculosis, but the record says nothing as to a possible presence of air vesicles.

DEFINITION AND DIFFERENTIATION OF THE DISEASE

Under this disease are included all the cases of spontaneous pneumothorax occurring in healthy persons without any demonstrable cause, in whom no signs of tuberculosis can be demonstrated — neither by auscultation and X-ray examination nor by bacteriological examination of sputum — and in whom the disease takes an afebrile course without any pleural effusion. Also such cases are included as show a slight rise of temperature in the first week of illness, and likewise the cases in which the X-ray plates show an insignificant exudate in the phrenocostal groove, too small to be made out on auscultation.

FREQUENCY, OCCURRENCE AND LOCALISATION

The lesion must be said decidedly to be rare. Out of the 51 patients, 36 were men, and 15 women. The lesion is most frequent in the age-class 20—40 years; and in this material it occurs about twice as often on the right side as on the left. In my 51 patients, 12 give some findings suggestive of cicatricial changes on the surface of the lungs. The Pirquet test has been made on 22 patients, with negative results in 9 cases.

SYMPTOMATOLOGY

In the great majority of these cases the disease sets in quite suddenly and, as a rule, without any accidental cause. Only in a minority of the cases does the lesion appear in connection with physical exertion.

In my material, all the different degrees of the lesion are represented, from the mildest to the most severe. According to the volume and pressure of the pneumothorax

air, the various types of the lesion are set up, and a description is given of their clinical features and course:

1. *Partial pneumothorax* (6 cases in my material).
2. *Coat-formed pneumothorax* (3 cases).
3. *Total pneumothorax without — or with slight — displacement* (22 cases).
4. *Total pneumothorax with moderate displacement* (14 cases).
5. *Tension pneumothorax* (10 cases).

Even though there are gradual transitions between these different types of the lesion, yet the cases of each group are showing a rather marked uniformity in their clinical features and in the course of the disease.

These types do not only represent the different degrees of the lesion, but they may equally well be taken as representing different stages of the disease, as some cases go through the whole development from a mild initial stage to a serious morbid condition.

Characteristic of the great majority of the pneumothorax cases is their clinical course as moderate and benign attacks of pneumothorax. This is rather peculiar in view of the fact that the disease is due to the rupture of a valve vesicle, and that it is therefore warrantable to assume that this rupture always results in a valve pneumothorax.

The most frequent forms are the total pneumothorax without — or with slight — displacement and the total pneumothorax with moderate displacement. The mildest degrees of the lesion — the partial pneumothorax and the coat-formed pneumothorax are observed but infrequently, probably because the diagnosis of such a mild attack may be made with certainty only by means of X-ray examination.

Next, an account is given of cases taking an unusual course. The various forms are:

6. *Chronic pneumothorax* (4 cases in my material).
7. *Hæmopneumothorax* (1 case).

8. *Recurrent pneumothorax* (7 cases).
9. *Alternating pneumothorax* (1 case).
10. *Bilateral pneumothorax* (not observed in my material).
11. *Pneumothorax alternating with Pneumopericardium* (not observed in my material).
12. *Pneumothorax with Subcutaneous Emphysema* (2 cases).

Of these forms, the recurrent pneumothorax is the most frequent. One or several relapses occur in about 20 per cent. of the cases of spontaneous pneumothorax in apparently healthy persons. In over half of these the relapse comes within one year after the first attack.

The term "alternating pneumothorax" applies to cases with pneumothorax on both sides but at different times. In more than one half of the cases reported so far, an attack on one side was followed within one year by an attack on the other side.

DIAGNOSIS

The diagnostic difficulties encountered in various forms of this disease are discussed in a separate chapter. The only symptom which is constantly present is the suppression of the respiration. But even this sign may sometimes —- in mild cases — be very hard to make out. In total pneumothorax without displacement, the usual tympanitic resonance may sometimes be absent, and the percussion note may be quite normal.

In some cases the localisation of the initial pains may be so misleading that they do not even suggest any lesion in the chest, and the illness may be regarded as lumbago or rheumatism of the shoulder. In my material there is one patient in whom a right-sided tension pneumothorax was taken to be a perforated gastric ulcer, wherefore laparotomy was performed. The diagnosis is often difficult in latent chronic pneumothorax. Right-sided hæmopneumothorax is often accompanied by tenderness and rigidity of

the upper part of the abdomen, so that the appearance of the case has a most deceptive resemblance to a case of perforated gastric ulcer. In every case the X-ray examination constitutes the chief diagnostic means.

PROGNOSIS

The outlook is good. The literature comprises only 6 cases wherein the patient have died from this disease. The cause of death was tension pneumothorax (1 case), hæmopneumothorax (4 cases), and bilateral pneumothorax (1 case).

None of my 51 patients have died from their spontaneous pneumothorax. The disease has a pronounced tendency to spontaneous recovery.

In most of the cases the air is absorbed within 6 weeks to 2½ months. In 4 of my cases the disease has been chronic, and has persisted for years; 3 of these have terminated in spontaneous recovery. Only in one of my cases does the condition of pneumothorax appear to be permanent, having persisted, presumably, about 20 years; and even in this case the condition is improving, the pneumothorax being now (September, 1931) merely partial.

The subsequent fate and condition of health of the patients have been ascertained by systematic after-examinations with Roentgenography, with the exception of two who could not be found. *In the 49 patients on whom the data are complete, only one has subsequently acquired pulmonary tuberculosis, evidently a later infection. Except in the two latest cases, the observation period for every after-examined patient has been at least 3 years.*

THERAPY

In *partial* and *coat-formed pneumothorax* the treatment is simply rest in bed for 2 weeks. In cases of *total pneumothorax* the patients must stay in bed at least 2 weeks; after this, the course of the disease may be shortened by

cautious aspiration of the pneumothorax air — but this should never be done till at least 2 weeks after the onset.

The slightest sign of *tension pneumothorax* is an indication for immediate puncture. Besides, it is essential to quiet the patient with morphine. It may be necessary to repeat the puncture.

In *chronic pneumothorax* the final outcome is often spontaneous recovery, even after the condition has been stationary for a number of years. One may try to cure a chronic pneumothorax with aspirations or, if this does not help, with intrapleural injection of 30 c.c. of a 30 % glucose solution. If necessary, the injections may be repeated.

The injection treatment ought to be employed in cases with *repeated relapses* and in *repeatedly alternating pneumothorax.*

In *hæmopneumothorax* one must realise that to move the patient implies a very grave risk. The treatment consists in rest and morphine. In desperate cases one may consider the possibility of an attempt with thoracotomy and ligation of the bleeding vessel.

In *bilateral pneumothorax* the treatment is bilateral puncture and permanent aspiration as described by Elte. The puncture is performed first on that side where the pneumothorax first set in, then on the other side. The aspiration is carried out by means of a water suction pump or an electrical suction pump, but it is not maintained longer than necessary to relieve the patient of his worst difficulties in breathing. If one is facing such a case without any accessories at one's disposal, an attempt must be made to save the patient by connecting the canule with a piece of rubber tubing and aspirating with one's own lungs. Of course, a large syringe or a rubber bulb may also prove serviceable in an emergency.

In *suffocating mediastinal emphysema with pneumothorax* the treatment consists in repeated punctures, oxygen inhalation and stimulation. It may be necessary to make a

small vertical incision in the skin over the jugulum and express the subcutaneous air by stroking.

Spontaneous pneumothorax in apparently healthy persons does not require any after-treatment whatever and, consequently, the patients should not be sent to a tuberculosis sanatorium. When, as demonstrated with this material, the patients later in life are doing so well as to health and working capacity, they should be spared the loss of time and the expenses of a sanatorium treatment and the fear of pulmonary tuberculosis.

The more important points of view derived from these studies may be summed up in the following

CONCLUSIONS

1. *"Spontaneous pneumothorax in the apparently healthy" is a nosographical category of practical value.*
2. *This term covers all cases of spontaneous pneumothorax that makes its appearance without any demonstrable cause in healthy persons, in whom no sign of tuberculosis may be demonstrated — neither by auscultation and X-ray examination, nor by bacteriological examination of sputum — and in whom the disease takes an afebrile course, without any pleural effusion. In this class are also included cases of this description with a slight rise of temperature in the first week of illness, and cases in which the X-ray plates show the presence of an insignificant pleural exudate, too small to be made out on auscultation.*
3. *The disease is subject to great variation in clinical features and course, and yet there are all degrees of transitions between the various types of the disease.*
4. *The disease is due to the rupture of a valve vesicle on the surface of the lung. There are two forms of valve vesicles. One form, the scar-tissue vesicles, develops*

especially in the apices of the lungs, next to a scar that may originate from a small healed tuberculous process. These scar-tissue vesicles are often multiple. The other form, the emphysematous valve vesicles, is produced alone through local emphysematous changes without scar tissue. These may be found as large solitary vesicles on the margins of the lungs. Probably the scar-tissue vesicles constitute the more frequent form of valve vesicles.

5. *Systematic after-examinations of a large number of patients with long observation period have shown that in practice it may be taken for granted that this disease has nothing to do with active tuberculosis. So these patients should be spared the loss of time and the expenses of a sanatorium treatment as well as the fear of pulmonary tuberculosis.*

For the sake of brevity, the writer ventures to suggest that *Spontaneous pneumothorax in the apparently healthy* be called *Pneumothorax simplex.*

RÉSUMÉ

Der findes i Literaturen offentliggjort ialt ca. 200 Tilfælde af Spontanpneumothorax hos tilsyneladende raske Mennesker, men i Opfattelsen af dette Sygdomsbillede har der ikke været Enighed mellem de forskellige Forfattere.

Paa Grundlag af et Materiale paa 51 Patienter med ialt 61 Pneumothoraxanfald gives her et Bidrag til Sygdommens Ætiologi, Symptomatologi, Diagnose, Prognose og Behandling.

Ætiologi og Pathogenese: Der foreligger i Literaturen ialt 6 Tilfælde, som er blevet anatomisk fuldt opklarede ved Autopsi. Undersøgelsen viste, at Aarsagen til disse Tilfælde har været en Bristning af overfladiske Blærer i Lungespidserne hos Mennesker med iøvrigt sunde Lunger. *Hayashi* og *Fischer,* som har beskrevet 3 af disse Tilfælde, har paavist, at disse isolerede Lungeblærer var dannet paa Grundlag af aragtige Forandringer i Lungespidserne, og Mikroskopien viste, at der ved Basis af disse Blærer fandtes en ventilagtig Klapdannelse, som bevirker, at disse Blærer i Tidens Løb udspiles mere og mere og tilsidst brister.

Sygdommen skulde derfor efter *Fischers* Opfattelse skyldes en Bristning af en saadan Ventilblære, dannet paa Basis af en lille Cicatrice i Lungespidsen.

Mine egne Undersøgelser over 3 Tilfælde af Ventilblærer, fundne som tilfældige Autopsifund, viser, at der findes to med Hensyn til Oprindelse og Bygning vidt forskellige Former for Ventilblærer, som begge ved deres Brist-

ning maa kunne give Anledning til Spontanpneumothorax.

Begge Former opstaar, fordi der ved Basis af Blærerne findes en Klapdannelse, men denne dannedes i det første af mine Tilfælde alene ved lokale emfysematiske Forandringer, og det lykkedes her at paavise Ventilen ved Mikroskopi. I det andet af mine Tilfælde fandtes flere Ventilblærer, opstaaet som Følge af et skrumpende Arvæv efter en lille udhelet Tuberkulose i Lungespidsen. I det tredie Tilfælde fandtes en solitær Arvævsblære i Lungespidsen, og det lykkedes ogsaa her at paavise en Klapventil ved Basis.

Der findes altsaa Ventilblærer baade med og uden Arvæv, hvilket ogsaa *Hayashi* tidligere har iagttaget.

Ved Røntgenundersøgelserne har det været muligt at paavise Ventilblæren paa Røntgenbilledet i eet Tilfælde (Nr. 42 *c*) i mit Materiale. Her præsenterer den sig som en hønseægstor Blære ved nederste Rand af højre Lunges nederste Lap. At Blæren kunde ses paa Røntgenbilledet maa dels skyldes, at Væggen i dette enestaaende Tilfælde for Størstedelen har været fibrøst fortykket, dels at Blæren var tilstrækkelig stor og havde en saadan Beliggenhed, at den ragede frit frem fra Lungeoverfladen. Patienten har ialt haft 3 Anfald af højresidig Spontanpneumothorax.

Resultatet bliver da:

Sygdommen skyldes en Bristning af en Ventilblære paa Lungeoverfladen. Der findes to Former af Ventilblærer. Den ene Form, Arvævsblærerne, opstaar især i Lungespidserne ved Siden af Cicatricer, som kan skyldes en udhelet Tuberkulose. Disse Arvævsblærer er ofte multiple. Den anden Form, de emfysematiske Ventilblærer, dannes alene ved lokale emfysematiske Forandringer uden Arvæv og kan findes som store solitære Blærer ved Lungerandene. Arvævsblærerne er sandsynligvis den hyppigste Form for Ventilblærer.

Hos een af mine Patienter foreligger senere Autopsi. Der fandtes ingen makroskopiske Tegn paa Tuberkulose. Sek-

tionsbeskrivelsen indeholder intet om eventuel Tilstedeværelse af Lungeblærer.

Sygdomsbilledets Definition og Afgrænsning: Til dette Sygdomsbillede regnes alle de Tilfælde af Spontanpneumothorax, som indtræder hos raske Mennesker uden paaviselig Aarsag, hos hvem der ikke kan paavises Tegn til Tuberkulose, hverken ved Stethoskopi, Røntgenundersøgelse eller Expectoratundersøgelse, og hvor Sygdommen forløber afebrilt og uden Exsudatdannelse. Hertil medregnes ogsaa Tilfælde, der viser en mindre Temperaturforhøjelse i Sygdommens første Uge og ligeledes Tilfælde, hvor der paa Røntgenbilledet ses et ubetydeligt Exsudat, som er saa lille, at det ikke kan paavises ved Stethoskopi.

Sygdommens Hyppighed, Forekomst og Lokalisation: Sygdommen maa absolut kaldes sjælden. Af de 51 Patienter var de 36 Mænd og 15 Kvinder. Sygdommen er hyppigst i Alderen 20—40 Aar. Højre Lunge var omtrent dobbelt saa hyppigt angrebet som venstre.

Af mine 51 Patienter er der 12, hvor man har Holdepunkter for, at der findes cicatricielle Forandringer paa Lungeoverfladen. Hos de 22 Patienter, hvor *Pirquets* Reaktion er udført, var denne i de 9 Tilfælde negativ.

Symptomatologi: Sygdommen opstaar i det overvejende Antal Tilfælde ganske pludselig og som Regel uden nogen Lejlighedsaarsag. Kun i et Mindretal af Tilfælde indtræder Sygdommen i Tilslutning til legemlige Anstrængelser.

I mit Materiale er alle Sygdommens forskellige Grader repræsenteret, lige fra de letteste til de sværeste. Efter Pneumothoraxluftens Mængde og Tryk beskrives de forskellige Typer af Sygdomsbilledet og deres Forløb:

1) *Den partielle Pneumothorax* (heraf findes i mit Materiale 6 Tilfælde).
2) *Den kappeformede Pneumothorax* (3 Tilfælde).
3) *Totalpneumothorax uden, eller med ringe Forskydning* (22 Tilfælde).

4) *Totalpneumothorax med middel Forskydning* (14 Tilfælde).
5) *Spændingspneumothorax* (10 Tilfælde).

Selv om der findes ganske jævne Overgange mellem disse forskellige Sygdomstyper, viser der sig indenfor disse Grupper en betydelig Ensartethed i det kliniske Billede og i Forløbet.

Disse Sygdomstyper repræsenterer ikke alene de forskellige Grader af Sygdommen, men maa i lige saa høj Grad betragtes som et Udtryk for Sygdommens forskellige Stadier, idet nogle Tilfælde gennemløber hele denne Udvikling fra et lettere til et sværere Tilfælde.

Det karakteristiske for det store Flertal af disse Pneumothorax Tilfælde er, at det er moderate og godartede Pneumothoraxanfald. Dette er ret ejendommeligt i Betragtning af, at Sygdommen skyldes en Bristning af en Ventilblære, og at man derfor maa gaa ud fra, at der altid dannes en Ventilpneumothorax.

De hyppigste Former er Totalpneumothorax uden, eller med ringe Forskydning og Totalpneumothorax med middel Forskydning. Sjældnere ses en Spændingspneumothorax. De letteste Grader: den partielle og den kappeformede Pneumothorax iagttages kun sjældent, hvilket rimeligvis skyldes, at Diagnosen af disse lette Tilfælde kun kan stilles med Sikkerhed ved Hjælp af Røntgenundersøgelse.

Der gives dernæst en Fremstilling af de sjældnere Former af Sygdommen. Disse er:

6) *Kronisk Pneumothorax,* hvoraf meddeles 4 Tilfælde.
7) *Hæmopneumothorax* (1 Tilfælde).
8) *Recidiverende Pneumothorax* (7 Tilfælde).
9) *Alternerende Pneumothorax* (1 Tilfælde).
10) *Dobbeltsidig Pneumothorax* (ikke iagttaget i mit Materiale).
11) *Pneumothorax alternerende med Pneumopericardium* (ikke iagttaget i mit Materiale).
12) *Pneumothorax med subcutant Emfysem* (2 Tilfælde).

Af disse er den recidiverende Pneumothorax den hyppigste. I ca. 20 % af Tilfældene af Spontanpneumothorax hos tilsyneladende Raske indtræder eet eller flere Recidiver. I over Halvdelen af disse indtræffer Recidivet inden et Aar efter første Anfald.

Den alternerende Pneumothorax kaldes de Tilfælde, hvor der kommer Anfald af Pneumothorax baade paa højre og venstre Side, men til forskellig Tid. Anfaldet i den anden Side indtraf i over Halvdelen af de hidtil kendte Tilfælde, inden der var gaaet et Aar efter første Anfald.

I et særligt Kapitel fremstilles den ofte vanskelige *Diagnostik* af Sygdommens forskellige Former. Det eneste Symptom, som er konstant til Stede, er den svækkede Respiration, men denne er undertiden ikke særlig udtalt i de lettere Tilfælde. Ved en Totalpneumothorax uden Forskydning mangler Tympanismen undertiden, og Perkussionstonen kan være ganske normal.

Initialsmerternes Lokalisation kan i nogle Tilfælde være saa vildledende, at de ikke viser hen til en Lidelse i Brysthulen, men Sygdommen opfattes som Lumbago eller Rheumatisme i Skulderen. I mit Materiale findes en Patient, hos hvem en højresidig Spændingspneumothorax blev antaget for et perforeret Mavesaar, hvorfor der blev gjort Laparotomi. Vanskelig er Diagnosen ofte ved den latente kroniske Pneumothorax. Ved en højresidig Hæmopneumothorax er der hyppigt Ømhed og Defence af den øverste Del af Underlivet, hvorfor dette Sygdomsbillede skuffende kan ligne et perforeret Mavesaar. Røntgenundersøgelsen er i alle Tilfælde det suveræne diagnotiske Middel.

Prognosen er god. I Literaturen findes kun meddelt 6 Tilfælde, hvor Patienterne er døde af denne Sygdom. Dødsaarsagen var Spændingspneumothorax (1 Tilfælde), Hæmopneumothorax (4 Tilfælde) og Dobbeltsidig Pneumothorax (1 Tilfælde).

Ingen af mine 51 Patienter er døde af deres Spontanpneumothorax. Sygdommen har en udpræget Tendens til Spontanhelbredelse.

Hos de fleste resorberes Luften i Løbet af 1½ til 2½ Maaned. 4 af mine Tilfælde har været kroniske med aarelangt Forløb; 3 af disse er endt med Spontanhelbredelse. Kun een af mine Patienter har en blivende Pneumothorax, som formentlig nu er ca. 20 Aar gammel, men denne kroniske Pneumothorax er nu (Septbr. 1931) kun partiel.

Ved systematiske Efterundersøgelser med Røntgenfotograferinger af Patienterne er disses senere Skæbne og Sundhedstilstand blevet oplyst. 2 af Patienterne kunde ikke findes ved Efterundersøgelserne.

Af de øvrige 49 Patienter, hvis senere Skæbne og Sundhedstilstand er fuldt oplyst, har kun een eneste senere faaet Lungetuberkulose. Særlige Forhold taler for, at dette ene Tilfælde skyldes en senere Infektion. Med Undtagelse af de to sidste har alle de efterundersøgte Patienter en Observationstid paa mindst 3 Aar.

Therapien bestaar ved *den partielle* og *den kappeformede Pneumothorax* i Sengeleje i 14 Dage. Ved *Totalpneumothorax* skal Patienterne ligeledes ligge i 14 Dage, man kan derefter afkorte Sygdomsforløbet ved forsigtig Aspiration af Luften, men dette bør først gøres, naar der er gaaet mindst 2 Uger efter Sygdommens Begyndelse.

Ved mindste Tegn paa *Spændingspneumothorax* gøres straks Punctur. Desuden er det nødvendigt at berolige Patienterne med Morfin, og Puncturen maa eventuelt gentages.

Ved kronisk Pneumothorax kommer der ofte tilsidst en Spontanhelbredelse, efter at Tilstanden har været stationær i Aarevis.

Man kan forsøge at helbrede en kronisk Pneumothorax ved Aspirationer eller, hvis dette ikke hjælper, ved Injection i Pleura af 30 ccm. af en 30 % Glukoseopløsning. Disse Injectioner maa eventuelt gentages.

Injectionsbehandlingen bør ligeledes anvendes ved stadig gentagne *Recidiver* og ved gentagne *alternerende Pneumothoraxanfald.*

Ved *Hæmopneumothorax* maa man være klar over, at der er en meget alvorlig Risiko forbundet med at flytte disse Patienter. Behandlingen bestaar i Ro og Morfin. I fortvivlede Tilfælde kan man overveje Muligheden af at forsøge en Thoracotomi med Ligatur af det blødende Kar.

Dobbeltsidig Pneumothorax behandles med dobbeltsidig Punctur og permanent Aspiration, saaledes som *I. Elte* har angivet. Man puncterer først den Side, hvor Patientens Pneumothorax er ældst, dernæst den anden. Aspirationen foretages ved Hjælp af en Vandsugepumpe eller en elektrisk Sugepumpe, men vedligeholdes ikke længere end nødvendigt for at befri Patienten for hans værste Aandedrætsbesvær. Staar man ganske uden Hjælpemidler, maa man gøre det Forsøg at sætte en Gummislange paa Kanylen og aspirere med sine egne Lunger. En stor Sprøjte eller eventuelt en Gummiballon vil naturligvis ogsaa kunne anvendes i Nødstilfælde.

Ved *suffocerende Mediastinalemfysem med Pneumothorax* foretages gentagne Puncturer, Iltinhalation og Stimulation, eventuelt lægges en lille lodret Incision i Huden i Jugulum, og Luften udpresses ved Strygning af Huden.

Ved Spontanpneumothorax hos tilsyneladende raske Mennesker er en Efterbehandling af Patienterne ganske unødvendig, og Patienterne bør derfor ikke sendes paa Tuberkulosesanatorium. Da det, som paavist i dette Arbejde gaar Patienterne saa godt senere i Livet, bør man spare dem saavel for et Sanatorieophold som for Ængstelsen for Lungetuberkulose.

De vigtigste af de Synspunkter, som er fremgaaet af dette Arbejde kan samles i følgende:

KONKLUSIONER

1. *Spontanpneumothorax hos tilsyneladende raske Mennesker er et klinisk Sygdomsbegreb af praktisk Værdi.*
2. *Til dette Sygdomsbillede regnes alle Tilfælde af Spontanpneumothorax, som indtræder hos raske Mennesker*

uden paaviselig Aarsag, hos hvem der ikke kan paavises Tegn til Tuberkulose, hverken ved Stethoskopi, Røntgenundersøgelse eller Expectoratundersøgelse, og hvor Sygdommen forløber afebrilt og uden Exsudatdannelse. Hertil medregnes ogsaa Tilfælde, som viser en mindre Temperaturforhøjelse i Sygdommens første Uge, og ligeledes Tilfælde, hvor der paa Røntgenbilledet ses et ubetydeligt Exsudat, som er saa lille, at det ikke kan paavises ved Stethoskopi.

3. *Sygdomsbilledet kan fremtræde under en Række forskellige Former, men saaledes, at der findes alle Overgange mellem de enkelte Sygdomstyper.*
4. *Sygdommen skyldes en Bristning af en Ventilblære paa Lungeoverfladen. Der findes to Former af Ventilblærer. Den ene Form, Arvævsblærerne, opstaar især i Lungespidserne ved Siden af Cicatricer, som kan skyldes en lille udhelet Tuberkulose. Disse Arvævsblærer er ofte multiple. Den anden Form, de emfysematiske Ventilblærer, dannes alene ved lokale emfysematiske Forandringer uden Arvæv og kan findes som store solitære Blærer ved Lungerandene. Arvævsblærerne er sandsynligvis den hyppigste Form for Ventilblærer.*
5. *Systematiske Efterundersøgelser af et stort Antal Patienter med lang Observationstid har vist, at man i Praksis kan gaa ud fra, at Sygdommen ikke har noget med aktiv Tuberkulose at gøre, hvorfor Patienterne bør spares saavel for et Sanatorieophold som for Ængstelsen for Lungetuberkulose.*

Som en kort Benævnelse af denne karakteristiske Sygdom vil Forfatteren foreslaa, at *Spontanpneumothorax hos tilsyneladende raske Mennesker* kaldes *Pneumothorax simplex.*

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CASE RECORDS

Abbreviations used in the case records:

A_I = First aortic sound.
A_{II} = Second aortic sound.
adm. = Admission to the hospital, admitted.
ausc. = Auscultation.
C. = Costa, rib.
disch. = Discharge from the hospital, discharged.
expect. = Expectorate.
IC. = Intercostal space.
P_I = First pulmonary sound.
P_{II} = Second pulmonary sound.
P. = Pulse.
perc. = Percussion.
phys. = Physical.
pr. = Pressure.
pt. = Patient.
resp. = Respiration, respiratory.
tp. = Temperature.

CASE 1.

Male, 43 years, official.

Kommunehosp. Dep. II, May 29, 1909—July 13, 1909.

No family history of tuberculosis. Past history of good health.

2 weeks ago sudden sharp pain in left side of chest, with some dyspnea. The family physician made a diagnosis of mild pleurisy. The stitch subsided in a couple of days, but was followed by a sensation of tension in epigastrium and left side, with increase of dyspnea on exertion. Pt. felt fairly well the first days and minded his work — made even political campaign speeches in evenings, although with great strain. Last 5 days he had to stay in bed for increasing dyspnea. His physician claims to have heard friction sounds in left side. Tp. normal at home.

On adm.: Left pneumothorax with diffuse distension effacing intercostal spaces. Resonant percussion note masking the heart-dullness, which extends from right sternal border to a little within right nipple. Resp. sounds faint, amphoric on coughing; a few times metallic tinkling. No succusion. Post. lung border at IC. X; tympanitic note below C. V. Right lung normal.

May 30. No cyanosis. P. 84.

May 31. 37.7/37.4. Feels better. P. 84; Resp. 16—18.

June 1. 37.5/37.4. *Puncture* in left mid-axillary line; *aspiration of 400 cc. of air.*

Tp. perfectly normal throughout stay in hospital.

June 2. No pain; some tension over chest. Circumference at level of nipples 89.5 cm.

June 3. Circumf. of chest 90.5; P. 72; Resp. 16.

June 5. *Puncture;*

aspiration of 4—500 cc. of air. After this, distinct heart-dullness with left border at left parasternal line.

June 15. *X-ray exam.:* No definite pneumothorax. Heart slightly displaced to right. Lung tissue condensed on lower right side.

July 1. Up.

July 6. Ausc.: Resp. a little distant on left side.

July 13. Pirquet: ÷. Discharged.

After-exam. April 27, 1928: Well during past 18¾ years. 1½ year ago, slight attack of broncho-pneumonia on left side with slight exudate. Recovery after 1 week in bed. *Ausc.:* Slight dullnes and slightly weaker resp. sounds on lower left post. surface. No other abnormalities. *X-ray exam.:* No abnormalities (Dr. *V. Finsen*).

Summary: Acute onset of left-sided pneumothorax with moderate displacement. Admitted to hospital 2 weeks later. Treated with 2 aspirations, with good effect. The air has disappeared 1 month later. After-exam.: Well during the past 18¾ years. No sign of tuberculosis.

CASE 2.

Female, 20 years, servant.

Kommunehosp., Dep. III, April 20—July 1, 1910.

No tuberculosis in family. Croup and scarlet fever in childhood. Otherwise always well, except in summer of 1909 when in 2—3 months she lost 7½ kg. and felt tired; got well again the next winter.

The present illness began quite *suddenly this morning* without preceding indisposition. She got violent pains in right side of chest and under scapula, and the pains were aggravated by deep resp. Vomited once. No cough or expectoration.

On adm.: Pt. is thin and pale. Tp. 38.1; P. 100. *Ausc.;* Almost no resp. motion of right half of chest. Resonant perc. note. No dullness. No râles. Resp. sounds weakened at base. No other abnormal sounds. Left lung normal.

Heart: P_{II} accentuated; otherwise normal findings. Abdomen slightly retracted. Liver at curvature.

April 21. 37.8/37.8. Pt. looks somewhat distressed. Continuous pains in right side, beginning a little below right breast and extending to right shoulder joint. Intercostal neuralgia?

April 22. 37.8/37.1. Steady pain, most on deep resp.. *Ausc.:* Liver dullness reduced to a narrow strip along the curvature. Resonant tympanitic perc. note on right side of chest; ant. surf., faint resp.; post. surf., faint bronchial resp. with amphoric touch and accompanied by faint metallic tinkling.

April 23. 37.4/37.2. *Tp. keeps perfectly normal throughout stay in hosp.* No cough or expectoration. *Ausc.:* resonant perc. note on right half of chest reaches ant. to C.VIII, post. to C.XII. No distension of the side.

April 29. *X-ray exam.:* Right lung compressed against hilus to size of a child's hand. Heart displaced but relatively little.

May 2. *Ausc.:* No particular changes.

May 7. *Pirquet:* +. Up.

May 13. *Ausc.:* Resp. a little faint on right side; otherwise about normal findings. *X-ray exam.:* Almost no more air in pleura.

May 20. *Ausc.:* Over right apex, fine crackling after coughing.

May 25. *X-ray exam.:* The lung appears fully extended (with exception of apex?). Right hilus shadow contains one pea-sized calcified focus and several smaller ones.

June 16. Feels perfectly well.

July 1. Discharged (transferred to sanatorium).

After-exam. is not made, as the patient could not be found.

Summary: Acute onset of right-sided pneumothorax with slight displacement. Admitted on the same day. At first taken to be intercostal neuralgia. The air is absorbed

spontaneously in a little over 1 month. No after-examination.

CASE 3.

Female, 38 years, widow.

Kommunehosp., Dep. III, Dec. 15, 1911—Jan. 6, 1912.

No tuberculosis in family. *Scrofulous* in childhood. From 16' to 19' year, she had „glandl sores" on both hands; at the age of 16, she was operated for ostitis of right hand.

Pneumonia at the age of 12. Since then, no pulmonary disease till adm. to this dep., Feb. 4, 1911; treated for *Pneumonia crouposa sinistra totalis,* accompanied by icterus. Disch. April 25. 1911. Well since.

Present illness began *6 days ago* with stabbing pains in left side of chest. At first, the pains were not very intense. On evening of 2' day, the pains were very severe and cutting, particularly in the back, on resp.. She was therefore somewhat short of breath. No subjective febrila. Pt. went to bed at once, and has kept it since. The pains have been less intense the last days. Slight cough. No expect. *On adm.:* Looks natural. No glandular swelling of the neck. Tp. 38,1; P. 92. *Ausc.:* Left side lagging a little, perhaps. On left post. surf., perc. note dull tympanitic over entire infrascapular reg. Resp. much impaired. No friction sounds. Vocal phenomena weakened. Right lung normal. *Heart* lung-covered; heart-sounds distant, but clear.

On back of right hand, 3 scars, 1 after resection of 3' metacarpal bone; 1 scar on back of left hand.

Dec. 16, 37,4 (morning). No particular dyspnea. No stitch. No expect..

Dec. 17. 37,7/37,2. *Ausc.:* Definite amphoric resp. round the angle of left scapula. *Pirquet:* +.

Dec. 18. *X-ray exam.:* Pneumothorax of left side. A few adhesions.

Tp. reached 37,8 one evening, otherwise perfectly normal throughout stay in hospital.

Jan. 6. Discharged.

After-exam.: May 2. 1928. Well during past 16 years.
Ausc.: No definite abnormalities.
X-ray exam.: Signs of emphysema; but no sign of infiltrative processes, or abnormal shades (Dr. *P. Blinkenberg*).
Summary: Scrofula in childhood. Ostitis of metacarpus in youth. At age of 38, acute onset of left-sided pneumothorax without displacement. Adm. 6 days later. Disch. after 3 weeks treatment, before the air is absorbed. After-exam.: Well during past 16 years. No sign of tuberculosis.

CASE 4.

Female, 43 years, farmer's wife.
Treated by Chief Physician J. Ussing, Frederikssund, April 1913.
At age of 21, she was 1 month in Frederikssund Hosp. for pleurisy. Otherwise past history of good health.
Present illness began April 13 with stitch in right side of the chest, marked dyspnea and cough.
X-ray exam.: (April 16, 1913): Right-sided pneumothorax with complete collapse of right lung. Displacement of heart to left and overlapping of pneumothorax on left side.
She was perfectly afebrile, and kept the bed about 1 month. As pneumothorax persisted with several small exacerbations, suggestive of chronic valvular pneumothorax, she was next year referred to Dr. med. *H. J. Panner* for
X-ray exam.: (April 23. 1914): Practically the same conditions as last year.
She was gradually getting used to her pneumothorax, but, at years' interval, she had several similar attacks, always setting in with marked dyspnea, and sometimes accompanied of stitch in the chest. The last symptom is not constant, however. The attacks come on, when she is feeling perfectly well, and are sometimes associated with cough, but never with fever. She has never shown any sign of tuberculosis.
After-exam.: July 14, 1928. Pt. is now 58 years, well

and capable of work. *Ausc.:* Slight emphysema. Lung-borders C.VII/C.XI; faint resp. over lower right post. surf. No other abnormalities. *X-ray exam.:* Right costodiaphragmatic angle obliterated and replaced by an intense shadow, with concave upper border. Entire right lung field slightly hazy; there seems to be some retraction of right apex. These changes are probably resulting from a previous pleurisy; no sign of infiltrative processes, neither old nor recent ones (Dr. med. *Busch*).

Summary: At age of 21, pleurisy. At age of 43: Acute onset of right-sided pneumothorax, taking a chronical course for several years. Later, occasional mild attacks. After-exam.: Apart from these attacks, good health in past 15 years. X-ray exam. shows thickening of pleura on lower right side. No sign of tuberculosis.

CASE 5.

Female, 39 years, joiner's wife.

Kommunehosp., Dep. III, July 15, 1913—Sept. 19, 1913.

No tuberculosis in family. „Inflammation of chest" in childhood; no pulmonary symptoms since. In October 1912, she caught cold and had bronchitis. She did not stay in bed, but the cough persisted the whole winter. 6 children, the youngest 3 months old.

Present illness began *2 months ago,* with *sudden onset* of dyspnea one morning, and sensation of „something coming apart inside". Pains increasing the following days, and marked dyspnea the first month. The coughing subsided, on the other hand, and during the last month, there has been neither cough nor expect.. Pt. has been up all the time, and does not think she has had any fever. *Expect.:* ÷ T. B.

On adm.: Pt. of vigorous build, in good nutrition. No dyspnea, Tp. 37,8; P. 80; Resp. 24. *Ausc.:* Distinct lagging of right side on resp.. Distension of right post. surf.. Resonant perc. note over entire right side. Resp. faint over upper, and absent over lower portion. Nu dullness; slightly

shortened tone over left apex. Ictus a little outside the nipple line. *X-ray exam.:* spontaneous pneumothorax. Right lung compressed downwards as an almost circular formation as large as an ostrich egg, and very dense in its basal part. In addition, some free fluid in pleural cavity.

July 16. 37.7/36.8. Tallquist: 70 %. *Tp. perfectly normal during entire stay in hosp.*

Aug. 8. *Ausc.:* Both sides of chest move about alike.

Aug. 28. *Ausc.:* Resp. heard over entire right ant. surf., vesicular, without râles or friction sounds. On post. surf., slight dullness in scapular region, where resp. is almost inaudible and accompanied by friction sounds. In infrascapular reg. slightly resonant perc. note; resp. vesicular, a little fainter than on left side.

Aug. 29. *X-ray exam.:* No sign of pneumothorax. Veil over right apex; some mottling of right lower lobe.

Sept. 11. *Ausc.:* No dullness post.. A few friction sounds ant. around right C. V.

Sept. 19. Discharged.

After-exam., May 15, 1928: Well during the past 14¾ years.

Ausc.: No abnormalities.

X-ray exam.: Veiling of left lung field, increasing from clavicle to base. In IC.II a fairly sharply defined shadow of small pea-size, probably due to calcified primary lesion. No other sign of infiltrative processes. Right lung normal. (Dr. *V. Holm*).

Summary: Acute onset of right-sided pneumothorax with moderate displacement. Adm. 2 months after. No exudate demonstrable on phys. exam., but effusion shadow on X-ray plate. Afebrile course. The air absorbed within 1½ month. After-exam.: Well during past 14¾ years. No sign of tuberculosis.

CASE 6.

Female, 31 years.

Private Pt. of Dr. Aug. Hübschmann, Nov. 1913.

No tuberculosis in family. Past history of good health.

Prensent illness began November 13, in the evening, as a violent stitch in the back, while the pt. was in bed. She did not seek medical aid, but consulted a masseuse, who massaged her energetically. She was getting more and more short of breath, however, wherefore she consulted her physician, who ascertained an extensive left-sided spontaneous pneumothorax. The condition was perfectly afebrile.

Jan. 27, 1914. *X-ray exam.:* (Dr. med. *Panner*): Total pneumothorax of left side with marked displacement of heart and trachea toward right. No adhesions. No exudate.

She was then examined by Chief Physician *G. E. Permin:*

Jan. 29, 1914. *Intrapleural pressure: ÷ 6 and + 5 cm. H_2O;* after aspiration of 150 cc. of air: ÷ 10 and 0 cm. H_2O. Tp. was normal; and no sign of tuberculosis could be made out.

Jan. 13, 1915. *X-ray exam.* (Dr. med. *Panner*): Still extensive left-sided pneumothorax. The lung a little less compressed than the year before, and the displacement also a little less. Very small effusion shadow in lower sinus (cf. Fig. 23).

March 16, 1920. Exam. again by Chief Physician *G. E. Permin.* Pt. is married since last exam., has born one child and is again pregnant. Feels fairly well; has been less dyspneic in past years. No cough, expect., sweating or fever. *Ausc.:* Heart borders a little indistinct. Ictus a little outside the nipple. Left ant. surf. of chest retracted, slight dullness and faint resp. from apex to IC.II. Feeble resp. in left axilla. Left post. surf., moderate dullness over supraspinal reg., slight dullness over infraspinal reg., and moderate dullness over infrascapular reg.. Right lung, slight dullness and faint resp. over supraspinal reg., otherwise no abnormality.

March 16, 1920. *X-ray exam.:* Only a little pneumothorax of left side, otherwise a diffuse fairly dense shadow

over entire side, probably due to thickening of pleura (irritation from the air). No definite signs of tuberculosis.

(Brother of pt. had also spontaneous pneumothorax, in 1921, described in detail in a paper by Chief Phys. *Erik E. Faber*).

June 27, 1923. *X-ray exam.:* Quite the same findings as in 1920.

After-exam.: June 18, 1928: Pt. has been perfectly well since. Never any febrile periods, and never any sign of tuberculosis or pleurisy. She feels perfectly well, when she does not have to work too hard or move too fast.

Ausc.: Entire left half of chest is considerably retracted and stays behind on resp.. Heart markedly displaced to left, right heart border being 1 finger's width to left of left sternal border, and ictus is found in IC.V in ant. axillary line. On left ant. surf., moderate dullnes and bronchial resp. from apex to C.II. Below this dullness, clear perc. note and vesicular resp. in IC.III whereafter this clear reg. goes over into the heart dullness. Left post. surf., moderate dullness and bronchial resp. from apex to a little below spina, then clear perc. note and vesicular resp. to angulus. In left infrascapular reg., moderate dullness and markedly suppressed resp.. No râles or friction anywhere. Right lung normal.

X-ray exam.: Form of chest somewhat emphysematous with distinct lung markings in right side, where there is no sign whatever of heart shadow. Heart shadow pulled over to the left, so that heart and mediastinum (with clearness of trachea in upper portion) are completely to the left of midline (there is slight dextroconvex scoliosis). Entire left half of chest is somewhat retracted, intercostal spaces smaller than on right side. Heart borders and the diaphragm are not distinct, being covered by a rather dense darkening over lower half of left thorax. Over upper portion, denser darkening extending to a little below the clavicle and down along the mediastinal shadow. Downward and medially, this darkening is sharply defined against

a marked clearance, which subsides downward, and in which there is something suggestive of lung tissue. In addition, this clearance covers several smaller, rather sharply defined, partly confluating clearances. Neither the relatively clear part, nor the darkened part show any shadows suggestive of previous or present infiltrations. The whole picture is most suggestive of pleural thickenings up and down, and the clearances might well resemble remnants of pneumothorax (Dr. med. *H. J. Panner*).

Summary: Acute onset of left-sided pneumothorax with chronic course. Pneumothorax persisted for years, without changing and without giving the patient any particular discomfort. On exam. 6½ years later, the left side is found to be retracted, with the heart pulled to the left, and with condensed areas upward and downward. In the middle of the lung, a clearer area that is interpreted as an encapsulated pneumothorax. No sign of tuberculosis. The same conditions are found on after-exam. 14½ years after the onset of the lesion. During all these years, the pt. has been perfectly well, and gone through 2 parturitions. There has never been any sign of pleurisy or tuberculosis. It is difficult to say how the retraction of the left lung is to be explained; perhaps it may be due to a pleurisy, which the pt. has not noticed, perhaps it is resulting from the protracted compression of the lung.

CASE 7.

Male, 20 years, draftsman.

Bispebjerg Hosp., Dep. B, May 27, 1914—July 28, 1914.

No tuberculosis in family. Past history of good health.

6 days ago, sudden onset of dyspnea during gymnastics, and pt. had a sensation of „getting thicker in right side of chest". Later a little pain in right side of chest and increasing dyspnea. Pt. claims to be well in all other respects; he thinks that there was some rise of tp. the first days of the present illness, as he perspired freely at night.

On adm.: Pt. is thin and slim. He looks slightly cyanotic and speaks with difficulty on account of „lack of air". Tp. 37,5; P. 72; Resp. 28, very superficial. *Ausc.:* Resonant tympanitic perc. on right ant. surf. from apex to C.VII, and on right post. surf. to C.XI. Resp. much suppressed, without râles or friction. Amphoric resp. on ant. surf.. Heart is partially lung-covered, feeble ictus in IC.V in nipple line.

May 28. No pain. Less dyspnea. *Pirquet:* +. *Tp. perfectly normal throughout stay in hosp.*

May 29. *Ausc.:* Right half of chest somewhat distended. No râles or friction. No sign of effusion. Liver reaches 2 fingers below the curvature.

May 30. *X-ray exam.:* Total pneumothorax of right side. Heart slightly displaced. Distinct markening of left lung.

June 19. Up.

July 9. *X-ray exam.:* Moderate pneumothorax of right side, complete over the area corresponding with the apex, and for the rest extending like a coat over the entire right lung. Left hilus shadows large, one pea-sized shadow at level of left hilus.

July 28. Discharged (to the country).

After-exam., June 12, 1928: Pt.'s mother developed pulmonary tuberculosis in her last years, and died of this disease last year, 68 years old. *Pt. has been perfectly well the past 14 years.*

Ausc.: No abnormality.

X-ray exam.: No abnormality (Dr. med. *Panner*).

Summary: Acute onset of right-sided pneumothorax with slight displacement. Adm. 6 days after. The air is absorbed gradually, but a little air is still remaining 6 weeks later. After-exam.: Well during past 14 years, no sign of tuberculosis.

CASE 8.

Female, 24 years, teacher.

Bispebjerg Hosp., Dep. B, Sept. 23, 1915—Oct. 11, 1915.

A. No tuberculosis in family, but in childhood pt. was somewhat exposed to infection from a tuberculous seamstress. Well in childhood, apart from a mild case af *scrofula* at the age of 9 years. Disposed to colds. In past half year, while cramming for examination, pt. lost considerable weight (7 kg.) and is pale and over-worked. Some dry cough with a little tenacious expect.

One week ago, „spots in the throat", coryza and cough. Pt. felt febrile, but did not take to bed. This condition has subsided somewhat.

On adm., Pt. pale, rather thin and delicate, Tp. 38; slightly elevated evening tp. next 4 days, and then *perfectly normal tp. throughout stay in hospital.* Slight swelling and redness of fauces on adm..

Sept. 24. *Ausc.: Subcutaneous emphysema of right ant. surf. covering the supraclavicular fossa and extending down to C.II.* Over entire right ant. surf. resonant perc., slight metallic sound on perc.. Resp. feeble, distant, but audible throughout. Over both post. surf. scattered sibilating rhonchi. On right post. surf. resonant perc. *Diagnosis:* Right pneumothorax; right subcutaneous emphysema.

Sept. 25. Sahli: 65 %. *Pirquet:* ÷.

Sept. 27. Feels well, no pain in chest. Only slight cough. A little subcutan. crepitation is still felt over right clavicle.

Sept. 28. Emphysema disappeared.

Sept. 29. *X-ray exam.:* No sign of pneumothorax. There seems to be veil over both apices with fine spots. Hilus shadows on right side, perhaps a little enlarged.

Oct. 1. *Pirquet:* ÷.

Oct. 9. *Pirquet:* ÷.

Oct. 11. *Ausc.:* Normal findings. Discharged.

Again in Bispebjerg Hosp., Dep. B, Dec. 22, 1917—Jan. 21, 1918.

Since the last adm., a brother of pt. has had hemoptysis. Pt. has felt well the past 2 years, apart from fatigue and loss of weight (5 kg.) in last 2 months through overwork for examination; Yesterday pt. felt a sudden stitch in the

back, across the lower part of thorax, aggravated by cough and resp. Feeling well otherwise. *Ausc.:* Ant. surf. and apices normal. In right infrascapular reg., very little dullness and slightly suppressed vesicular resp. without râles or friction. The first evenings, tp. was slightly elevated (37,9—37,7), but was otherwise perfectly normal throughout stay in hospital.

Dec. 28, *Expect.:* ÷ T. B.

Jan. 10, *Ausc.:* Normal findings.

Jan. 21. Discharged.

B. *Pt. was then well the next 6¾ years till 1924, when she had a new right-sided pneumothorax, for which she was adm. to Vejlefjord Sanatorium, where she was treated Oct. 11, 1924—April 7, 1925.*

Abstract of record: *2 weeks ago, sudden onset* of severe pain in right side of chest and dyspnea. Pt. was up the first 4 days, but had to go to bed on account of pain and dyspnea. Diagnosis was made of extensive right-sided pneumothorax, and pt. went therefore from Copenhagen to Vejlefjord Sanatorium. She stood the trip well. *Ausc.:* Suppressed resp. over entire right lung. A few cracklings after cough; also occasional crackling over left apex. *X-ray exam.:* Pneumothorax of entire right side. Lung compressed to about one half. No exudate. No infiltrations of the lung tissue.

Oct. 14. Pressure in pneumothorax cavity: ÷ 2½ and + 1½ cm. H_2O.

Pt. treated with Sanatorium therapy, and with maintainance of pneumothorax; 7 air inflations while in Sanatorium. Tp. normal throughout this period. *Expect.:* ÷ T. B. (11 times).

Pt. went then to Copenhagen, and continued with *ambulatory pneumothorax treatment* at the same time as she returned to her school work (summer 1925); and she has practically not been ill one day since. She was inflated every 2—3 weeks, and her pneumothorax was kept up for

almost 2½ years till February 1927, whereafter the lung expanded. T. B. were never demonstrated in expect.

After-exam. March 14, 1929: Feels perfectly well. *Ausc.:* Slightly suppressed resp. over right lung; no other abnormality.

X-ray exam.: Rather intense veiling of entire right lung field, but with appearance of usual lung markings. No sign of infiltrative processes. No exudate. The changes resemble the results of pleurisy (Dr. *P. A. Blinkenberg*).

Summary: Scrofulous in childhood. At age of 24 years, mild bronchitis complicated by right-sided partial pneumothorax with subcutaneous emphysema around the clavicle. Air disappeared in 5 days. 2 years later, an attack of illness, which probably was a mild case of pneumothorax. 8¾ years after the first attack, acute right-sided total pneumothorax without displacement. Treated in tuberculosis sanatorium ½ year with maintainance of pneumothorax by means of air insufflations. Pneumothorax then kept up by ambulatory treatment for almost 2½ years. Otherwise perfectly well and capable of work. There has never been any definite sign of tuberculosis in past 13 years. After-exam.: Slight suppression of resp. and veiling of right lung. No sign of tuberculosis.

CASE 9.

Male, 24 years, warehouse worker.

Bispebjerg Hosp., Dep. C. July 1, 1917—July 20, 1917.

One sister has pulmonary tuberculosis. Pt. always well before.

5 days ago, pt. had to exert himself in carrying heavy loads. In the evening, on his return to home, he had *suddenly* a violent stitch in right side of chest and felt somewhat short of breath. He went to bed, and has stayed in bed since. Dyspnea disappeared already on day after onset. But the stitch persisted, and has been associated with marked palpitation. He does not think he has had any fever. No cough or expect.

On adm.: Healthy appearance, Tp. 37,6; P. 78; Resp. 20. *Ausc.:* Dullness over right apex. Tympanitic perc. note over rest of right lung, extending to C.VII and C.XII. Metallic tinkling over entire lung. No râles. No Hippocratic succussion splash. Left lung normal. Heart is displaced; right border 1 finger to left of left sternal border. Ictus in IC.VI in nipple line. Heart sounds clear.

July 3. *Expect.:* ÷ T. B.

July 7. *X-ray exam.:* Right-sided pneumothorax. Veil over upper half of left side, increasing upwards.

Tp. perfectly normal throughout stay in hosp.

July 10. *X-ray exam.:* Pneumothorax decreasing.

July 12. *Ausc.:* No changes. Up.

July 20. *Ausc.:* No abnormal dullness. Discharged.

Aug. 22. *X-ray exam.:* Pneumothorax disappeared.

After-exam.: May 10, 1928: Perfectly well during past 10¾ years. *Ausc.:* No abnormality. *X-ray exam.:* No sign of present or previous infiltrative processes; but suggestion of emphysema (Dr. med. *H. J. Panner*).

Summary: Acute onset of right-sided pneumothorax with moderate displacement. Air absorbed spontaneously in about 8 weeks. After-exam.: Well in past 10¾ years, no sign of tuberculosis.

CASE 10.

Male, 20 years, electrician.

Bispebjerg Hosp., Dep. B. Dec. 21, 1918—Jan. 19, 1919.

No tuberculosis in family. Past history of good health.

5 days ago, pt. awoke in the morning with a violent stitch in the chest, on which account he stayed in bed that day. As he felt better the next day, he got up, but had to go back to bed with pain in back an chest and some cough. Nu subjective febrilia.

On adm.: Pt. does not look sick. No cyanosis or dyspnea. Tp. 38; P. 100, regular, strong; Resp. 20. *Ausc.:* No dullness. Resp. suppressed bronchial on ant. and post. surf. of right side.

Dec. 22. 38/36,8. No distress.

Dec. 23. 37,6/37,1. *Ausc.:* Right half of chest a little distended, moving but little on resp. Right ant. surf., tympanitic perc. note from C.II to base. Right post. surf. tympanitic perc. note from a little below spina to base; over lower half marked amphoric metallic resp., and vocal sounds also markedly metallic. No râles, no succussion.

Dec. 24. 37,7/36,7. Persisting pain in right side on resp. and cough.

Tp. slightly elevated (37,8—37,9) a couple of evenings, then perfectly normal throughout stay in hosp.

Jan. 2. *Ausc.:* Normal. Up.

Jan. 11. *X-ray exam.:* Small area of pneumothorax on lower right side. Veiling of left apex and upper half of left side. Large hilus shadows on both sides.

Jan. 19. Discharged.

After-exam.: June 5, 1928: Perfectly well in past 9½ year. *Ausc.:* No abnormalities. *X-ray exam.:* No definite signs of veilings or of infiltrative processes (Dr. med. *Busch*).

Summary: Acute onset of right-sided pneumothorax without displacement. Adm. 5 days later. Air is absorbed rapidly; only a little remaining after 3 weeks. After-exam.: Well in past 9½ years, no sign of tuberculosis.

CASE 11.

Male, 25 years, clerk.

Kommunehosp., Dep. III, March 7, 1919—April 17, 1919.

Family history negative. Past history of good health, apart from mild attack of rheumatic fever 5 years ago.

This morning, when feeling perfectly well and on his way to work, pt. had a sudden attack of violent pains in entire right half of chest accompanied by shortness of breath. The pains were aggravated by resp.. About the same time, a little dry cough without expect..

On adm.: Pt. is cyanotic, congested to the face. Rather exhausted and distressed, suffering great pain on turning in bed, wherefore phys. exam. is restricted to head and chest. Tp. 37,5; P. 96, Resp. ca. 40. He lies on right side. *Ausc.:* Tympanitic perc. over entire right lung. No dullness. Entire right side is lagging under resp.. Marked suppression of resp. over entire right lung, with hollow metallic sound; Resp. inaudible in some places. Distinct metallic tinkling. No râles. Left lung normal. Ictus in ant. axillary line. $P_{II} > A_{II}$. Marked impetus. Rx: Morph. Chlor. 0,015. Stimulation with camphor and caffein injections.

March 8. 37,3/37,7. Repeated injection of morphin. Looks healthy, but cyanotic. P. 100. *Ausc.:* Ictus 2 cm. outside nipple line. Liver just below curvature. *During the day, increasing dyspnea and cyanosis. At 10 P. M., March 8, marked dyspnea and distress; Extreme displacement of heart (ictus in ant. axillary line). Therefore — in IC.IV in right axilla — Puncture,* with pneumothorax apparatus.

Initial pressure ..... + 13 and + 20 cm.
After aspiration of 1200 cc. of air ÷ 1 and + 5 cm.

Under aspiration, considerable decrease of dyspnea and cyanosis; afterwards ictus felt in nipple line.

March 9. 38,6/37,7.

March 10. 37,8/37,9. No shortness of breath when lying quiet. Appearance only slightly cyanotic. *Pirquet:* +. *Ausc.:* Loud tympanitic perc. note and suppressed resp. over entire right lung, ant. to C.VII, post. to C.XII. Metallic tinkling on coughing. Ictus in IC.V, 2 cm. outside the nipple line. Left lung normal. Liver reaches umbilical transversal.

March 12. *X-ray exam.:* Total pneumothorax of right side with displacement of heart over in left half of thorax, where the lung is very condensed from pressure. Trachea clear to left of the spine.

Tp. slightly elevated a few evenings (37,7—37,8), from March 17 perfectly normal.

March 13. Ictus reaches almost ant. axillary line.

March 17. Increasing displacement of heart.

Puncture

Initial pressure ..... + 2 and + 4 cm. H_2O

After aspiration of 400 cc., average pr. ca. ÷ 1 cm. H_2O.

Ictus has now moved a little over 2 cm. inward.

March 20. Feels well. *Expect.:* ÷ T. B. *Pirquet:* +.

March 25. Ictus 3 cm. outside nipple line.

March 27. Up.

April 8. *X-ray exam.:* Right lung compressed toward midline, so it forms a ca. 6 cm. wide zone parallel with the spine in lower half of lung field; its upper borders can not be made out. No effusion. Heart slightly displaced to left.

March 29. *X-ray exam.:* Now only slight amount of air in phrenicocostal sinus and in apex; lung distended elsewhere. Discharged.

After-exam., June 16, 1928: Perfectly well the past 9 years. *Ausc.:* Normal findings. *X-ray exam.:* Suggestion of pulmonary emphysema, but no sign of infiltrative processes (Dr. med. *H. J. Panner*).

Summary: Acute onset of right-sided tension pneumothorax. Adm. immediately in poor condition. Next evening the condition is critical, so puncture is performed. During the following days, again increasing displacement, for which puncture is repeated on 10' day. After this, the tension symptoms subside and the air is almost completely absorbed after 7 weeks. After-exam.: Well in past 9 years. No sign of tuberculosis.

CASE 12.

Male, 30 years, warehouse worker.

Kommunehosp. Dep. II. April 15, 1919—June 8, 1919.

A. No tuberculosis in family. Past history of good health.

At 3 A. M., sudden onset of pain in right side of chest, radiating out in back. The pains were continuous, and aggravated by coughing and deep resp. Later in the day, he

got short of breath, and has expect. a little tenacious mucus. This evening, pains in the precordium and marked palpitation.

On adm.: Pt. is a little pale, perspiring, somewhat dyspneic. Tp. 37,6; P. 84; Resp. 36. *Ausc.:* Suppressed resp. over entire right side. No dullness.

April 16. 37,6/37,5. P. 72, Resp. 20. He is pale, with slight cyanosis of the cheeks (small varices). *Ausc.:* Right ant. surf. distinctly protruding, moves less on resp. Slightly tympanitic perc. note. Lung-liver border at C.VII; on post. surf. at C.XII. Resp. suppressed; distinctly amphoric ring over infrascapularis. Ictus in IC.V, 1—2 fingers outside nipple line. Relative heart dullness on right side reaches to left sternal border.

Tp. normal throughout stay in hosp.

April 18. Getting very dyspneic on moving in bed. P. 68, Resp. 24.

April 20. *X-ray exam.:* Total pneumothorax of right side. The lung is compressed, so it occupies but half of the pleural cavity.

April 23. *Pirquet:* Doubtful.

April 24. *Expect.* ÷ T. B.

April 29. Less dyspneic.

May 22.*Ausc.:* Resp. still suppressed on ant. and post. surf.. No displacement of heart. Up.

June 4. *X-ray exam.:* No sign of pneumothorax.

June 8. *Ausc.:* Normal findings. Discharged.

B. *Treated for relapse, in same Dep., Dec. 10, 1919—Jan. 17, 1920.*

From the record: Pt. feeling well till *4 days ago,* when there was *sudden onset* of pains under right scapula. He was working till yesterday, however, when the pains were aggravated, and he became short of breath; also pains over precordium, accompanied by faintness. Some cough, very little expect..

On adm.: Pt. is pale, perspiring. Tp. 38,9. P. 108. Resp. 44. Right side appears somewhat distended and lagging on

resp. Slightly tympanitic perc. note. Faint resp. No râles. Right heart border 1 finger to left of left sternal border. Ictus 2 fingers outside nipple line. Heart action regular, clear sounds. Liver about 1 finger below curvature.

Dec. 11. 38,4/37,2. A little pale with cyanotic tinge. No dyspnea in quiet recline. P. 88, Resp. 26.

Dec. 12. 36,8/36,7. *Tp. perfectly normal during rest of stay in hosp.*

Dec. 30. Up.

Jan. 12. *Ausc.:* No dullness. Perhaps slight suppression of resp. over right lung. Scanty friction over lower right ant. surf. Expect. ÷ T. B. (2 times).

Jan. 15. Ictus now in IC.IV in nipple line.

Jan. 17. Discharged.

Subsequent history: Perfectly well for 3 years till beginning of January 1923, when he had acute lobar pneumonia. Was adm. to Kommunehospitalet Dep. II Jan. 5, and died Jan. 12 under increasing dyspnea and heart failure. Lobar pneumonia of left lower lobe. Right lung normal. Autopsy not permitted.

Summary: Acute onset of right-sided tension pneumothorax. Adm. at once. Air absorbed in 7 weeks. 8 months later, repeated attack of acute right-sided tension pneumothorax, developing in 4 days. Air disappearing gradually. Then perfectly well for 3 years till onset of left-sided lobar pneumonia with fatal issue.

CASE 13.

Male, 40 years, official.

Kommunehosp., Dep. III, May 26, 1919—May 27, 1919.

Family history negative. Past history of good health.

2 weeks ago, sudden onset of dyspnea in the night, so pt. awoke and had to sit up to get air. The condition has kept about the same, pt. still being short of breath, with sensation of pressure in left half of chest.

On adm.: Looks natural, Tp. 37,2, P. 88, Resp. 16. Left

half of chest distended, with tympanitic perc. note and inaudible resp. Right lung normal. Heart dullness can not be made out.

May 27. Tp. normal. *Ausc.:* Left lung border post. at C.XII. Distinct metallic tinkling after coughing. Resp. inaudible. *X-ray exam.:* Total pneumothorax of left side. Minimal effusion shadow in phrenicocostal sulcus. Heart markedly displaced to right. No definite sign of infiltration in right lung. No adhesions observed, except perhaps to medial half of diaphragm. Discharged.

After-exam.: May 2, 1928: Perfectly well past 8¾ years. *Ausc.:* No abnormality. *X-ray exam.:* No abnormality (Dr. med. *H. J. Panner*).

Summary: Acute onset of left-sided pneumothorax with displacement. Adm. 2 weeks later for observation. After-exam.: Well during past 8¾ years, no sign of tuberculosis.

CASE 14.

Male, 16 years, student.

Kommunehosp., Dep. III, March 9, 1920—March 23, 1920.

Family history negative. Past history of good health.

7 weeks ago, while sitting in the auditorium, pt. was *suddenly* taken very ill with cough and pains in right side of chest. His physician took it to be right-sided pleurisy. Stayed in bed for 2 weeks, and then at home for 1 month. Slight cough. Insignificant expect. (÷ T. B.). Has been troubled a good deal with shortness of breath when ascending stairways. Tp. is said to have been normal throughout the illness.

On adm.: Looks natural. Right half of chest considerably distended. Slight dullness over right apex, then tympanitic perc. note, and almost inaudible resp. to C.VI. On right post. surf., tympanitic perc. from apex to base at C.XII. Resp. suppressed over upper part, almost inaudible over

lower part. No succussion splash. No râles. Left lung normal. Heart findings normal. *Pirquet:* ÷.

Tp. perfectly normal throughout stay in hosp.

March 13. Feels well. *X-ray exam.:* Right-sided pneumothorax, lung compressed to $^2/_3$ of normal volume. No effusion. Some spots at left hilus. Heart is not seen to right of spine, and gives a narrow dropping-heart shadow.

March 17. *Pneumothorax insufflation.*

Initial pressure ÷ 2 + 2 cm. H_2O

After insufflation of 600 cc. of air + 3 + 8 cm. H_2O

March 18. Up.

March 23. Discharged (Cont. pneumothorax treatment).

After-exam.: Impracticable, as pt. lives abroad. In a letter dated Danzig, March 19, 1928, he says: „Since my illness, I have not been sick one day.". Thorough medical exam. in October 1926 and Aug. 1927; both times declared to be perfectly normal.

Summary: Acute onset of right-sided pneumothorax with moderate displacement. Adm. 7 weeks later. According to written communication, pt. has been perfectly well the past 8 years, and on medical exam. he has been declared perfectly healthy.

CASE 15.

Male, 34 years, painter.

Bispebjerg Hosp. Dep. B, September 18, 1920—Nov. 17, 1920.

Past history of good health.

1½ year ago, while feeling perfectly well, he suddenly noticed shortness of breath on exercise. Stayed in bed at home 3 weeks, with some fever; got up, but did not feel quite well, had pains in chest and under right scapula. Improved gradually, and has been able to work. Had 2 relapses with pains in chest and back; pains disappeared after rest for 8 days.

Last 8 days, again pain in right side of chest, and cough.

No expect. He thinks he has had no fever, but has been short of breath.

On adm.: Tp. 37,7, P. 92, Resp. 16. *Ausc.:* Over entire right lung, tympanitic perc. note with faint or inaudible resp.. No râles. Left lung normal.

Sept. 19. *X-ray exam.:* Large right-sided pneumothorax occupying lateral and lower half of lung field. Some displacement of upper part of heart to left. Calcified glands at left hilus.

Sept. 21. *Expect.:* ÷ T. B.

Apart from first day, tp. perfectly normal throughout stay in hosp.

Sept. 27. Feels well. Up.

Nov. 17. Transferred to sanatorium.

Treated in Boserup Sanatorium, Nov. 26, 1920— May 20, 1921.

Tp. normal. Expect. ÷ T. B.. Pneumothorax subsiding very slowly. On adm., the air occupied a triangular space in lower half of pleural cavity. On May 18, X-ray plate still showed a clearance of this area, about as large as a hen's egg. On discharge, ausc. showed persisting tympanitic perc. note, and faint resp. over entire right lung.

After-exam. June 1, 1928: Perfectly well in past 7½ years. *Ausc.:* No abnormality. *X-ray exam.:* Diffuse veiling of entire right side, especially downward; distinct lung markings made out through the veil, mostly streaked and probably originating from chiefly normal lung structures; but one can not exclude the possibility of a shadow over lower portion, most likely due to connective tissue formations. Hardly sign of any recent process. Left side normal. It may be that this is a case of results from an old tuberculosis (Dr. med. *H. J. Panner*).

Summary: Right-sided pneumothorax with slight displacement, probably several previous light attacks. Adm. 8 days later. Protracted course. Even after 8 months, the air has not disappeared completely. After-exam.: Perfectly

well the past 7½ years. X-ray exam. shows fibrous changes on lower right side. Ausc. shows no abnormality.

CASE 16.

Male, 41 years, compositor.

Kommunehosp., Dep. II, Nov. 19, 1920—Dec. 12, 1920.

1 sister treated in Finsen Institute for a „bump" on the neck. Otherwise no family disposition to tuberculosis. Past history of good health.

Last night, sudden onset of present illness. Pt. was slowly going downstairs, when he had a violent stitch in right side of chest, together with cough and a little expect. (blood-tinged). In addition, marked dyspnea, shivering, profuse sweating, faintness and anxiety. Tp. 37,3.

On adm.: Slept poorly last night. Complains mostly of very marked dyspnea and violent stitch under right scapula and in precordium and upper part of epigastrium. Face rather congested, slightly cyanotic. Very marked dyspnea. Resp. 32, superficial; Tp. 37,5; P. 96. *Ausc.:* Right half of chest somewhat distended, mowing less on resp.. Tympanitic perc. note. Resp. very faint, almost inaudible over right lung, bronchous, with peculiar distant metallic note. No dullness. No râles. On left side a few mumbling rhonchi. Ictus 1 finger to left of nipple line. $P._{II}$ accentuated.

Nov. 20. 37,5/37,6. P. 104; Resp. 36. Slight cyanosis. Facial expression anxious. *Ausc.:* Right side of chest still greatly distended, with effacement of intercostal spaces. Lower border on right side at IC.VII. Tympanism over sternum. Left heart border 2 fingers lateral to nipple line. Faint resp. with amphoric note. No exam. of post, surf. Liver 1 finger below the curvature. Bl. pr.: 130 mm. Hg.

Puncture,

with evacuation of 300 cc. of air, beside some air that escaped under connection of the tube.

Nov. 21. 37.5/37.1. Much improved after puncture.

From now on, tp. perfectly normal.
Nov. 23. Expect. ÷ T. B.
Nov. 25. P. 66, Resp. 24.
Nov. 30. *Pirquet:* +.
Dec. 11. *Ausc.:* Practically normal findings.
Dec. 12. Well. Discharged.
(No X-ray exam. of this pt.)

After-exam. June 11, 1928: 1 brother died of pulmonary tuberculosis. Pt. has been well during past years, apart from a protracted phlebitis with embolism of right lung, in 1925. *Ausc.:* Moderate emphysema, otherwise normal findings. *X-ray exam.:* Some emphysema, no sign of infiltrations in the lungs (Dr. *V. Holm*).

Summary: Acute onset of right-sided tension pneumothorax. Adm. next day. Following day, puncture with marked relief. After-exam.: Apart from phlebitis with pulmonary embolism, well during past 7½ years. No sign of tuberculosis. Moderate emphysema.

CASE 17.

Male, 32 years, bookkeeper.

Private pt. of the late Dr. L. Fr. Lunddahl and Dr. Svend L. Brorson, April 1922.

No tuberculosis in family. Past history of good health.

While on his way to his office in the morning, pt. had *suddenly* a violent stitch in right side of back. The stitch gave him some trouble, but he tended his work that day, and went out in the evening. Next day, he also went to work, but on account of increase of the stitch, and dyspnea, he had to quit work at noon. Much distressed, he consulted his physician, who at once made the diagnosis and sent him home to bed. He stayed in bed almost 4 weeks. The first days, he was very dyspneic, especially when lying flat on his back, but after this, he was feeling perfectly well. Tp. slightly elevated the first days, then perfectly normal. *X-ray exam.,* 24 days after onset of illness, showed remnants of

right-sided pneumothorax in phrenicocostal sinus; hilus shadow slightly enlarged on right side, with a few pea-sized calcifications. No other abnormalities. No exudate.

After-exam. May 8, 1928: Well during past 6 years. *Ausc.:* Slight extension of lung borders on post. surf.; no other abnormalities. *X-ray exam.:* No abnormality on left side. On right side, very dense lung markings in lower portion. and — particularly conspicuous in a few areas in IC.II ant. — small clearances, which *might* be bronchiectasis. No definite sign of tuberculosis in the lungs. (Dr. med. *P. Flemming Møller*).

Summary: Acute onset of right-sided pneumothorax, probably without displacement. Air almost disappeared after 24 days. After-exam.: Well during past 6 years. No sign of tuberculosis.

CASE 18.

Male, 26 years, agent.

Frederiksberg Hosp., Dep. B, May 9, 1922—June 12, 1922.

No tuberculosis in family. Past history of good health.

1 week ago, after a brisk walk in the evening, and having run a little, pt. had *suddenly* a stitch in right side of chest, radiating out in back. This was accompanied by some kind of „oppressive sensation" (not real anxiety), and sweating. Pains lasted most of the night, then subsided without remedies. Pt. has stayed in bed since, but feels perfectly well. No dyspnea or palpitation. No fever. His physician, Dr. *Axel Olufsen,* made the diagnosis pneumothorax.

On adm.: Looks natural. No dyspnea. Tp. 37,7: P. 88; Resp. 22. *Ausc.:* Right post. surf. distended and lagging. No dullness. Faint amphoric resp. over entire right side.

Resp. 24—28 during first days, then gradually slower, and 16 on discharge. Tp. 37,7 the first 2 evenings; *from then, perfectly normal tp. during entire stay in hosp.*

May 11, *X-ray exam.:* Total pneumothorax of right side.

Lung compressed to a shadow, ca. 6 × 14 cm., in lower part of field. Left lung normal.

May 29, *X-ray exam.:* Pneumothorax subsiding.

June 12. Discharged.

After-exam.: May 1, 1928: Perfectly well during past 6 years. *Ausc.:* Resonant perc. note over ant. surf. on both sides. Lung borders normal. Otherwise normal findings. *X-ray exam.:* No sign of infiltrative processes. There seems to be some emphysema (Dr. med. *H. J. Panner.*)

Summary: Acute onset of right-sided pneumothorax without displacement. Adm. 8 days later. Air is absorbed rapidly. After-exam.: Well during past 6 years, no sign of tuberculosis.

CASE 19.

Male, 29 years, sailor.

Bispebjerg Hosp., Dep. C, Dec. 28, 1922—Febr. 22, 1923.

No tuberculosis in family. Well in childhood and adolescence. 8 years ago, treated in hosp. in Egypt for left-sided exudative pleurisy; fluid was evacuated from pleural cavity. Since then, perfectly well.

1 month ago, when exerting himself in lifting a barrel, he felt a sudden stitch in chest and tension in left side. At first, he was very exhausted from pain and dyspnea. No fever. Has been up all the time. Lately, again troubled by dyspnea and palpitation.

On adm.: Ausc.: Left half of chest somewhat protruding. Resp. almost inaudible over entire left lung, except upwards, where it is bronchial. No dullness. No heart sounds are heard on left side of midline; but heart action heard distintcly to right of sternum, where there is some dullness going over in the liver dullness.

Dec. 29. *Electrocardiogram:* Normal findings.

Explor. Puncture:

Outlet of air coincident with expiration.

Tp. normal throughout stay in hosp.

Jan. 20. *X-ray exam.:* Extensive left-sided pneumothorax. Heart markedly displaced to right. Right lung shows calcifications in hilus shadow and some veiling, especially of lower part. Some infiltration in right apex.

Febr. 14. *X-ray exam.:* No sign of pneumothorax.

Febr. 22. Discharged.

After-exam. May 21. 1928: Perfectly well during past 5¼ years. *Ausc.:* Normal findings. *X-ray exam.:* On special plate of apices, moderate retraction of the lung tissue in left apex, where intercostal spaces seem smaller, perhaps indicating an old tuberculous process. Apart from this, no definite sign of infiltrative processes or abnormal veilings anywhere in lung fields (Dr. *V. Finsen*).

Summary: At age of 21 years, left-sided exudative pleurisy. 8 years later, acute onset of left-sided pneumothorax with displacement. Adm. 1 month later. Air is absorbed in about 1½ month. After-exam.: Well during past 5¼ years. X-ray exam.: Retraction of left apex. Ausc.: No abnormality.

CASE 20.

Male, 21 years, mechanic.

Øresundshosp., Med. Dep., January 9, 1923—March 20, 1923.

No tuberculosis in family. Past history of good health.

1 week ago, sudden onset of stitch in right side of chest, accompanied by dyspnea. No palpitation; no cough or expect.. No fever. Stayed in bed 3 days, whereafter pt. has been up, feeling well, and not troubled by dyspnea. Stitch disappeared.

On adm.: Looks natural. Tp. 37,1; P. 72; Resp. 16. *Ausc.:* Faint resp. over entire right side of chest; no other particular abnormalities of lungs. Right heart border at left sternal border. Ictus not palpable.

Tp. perfectly normal throughout stay in hosp.

Jan. 13. *X-ray exam.:* Diffuse veiling of left lung, with

several small compact areas between clavicle and C.II. Right lung field: Large air-filled space, lung occupying but lower medial half of chest cavity; small exudate.

Jan. 15. *Pneumothorax insufflation* on right side. Initial pressure ÷ 8 and ÷ 2 cm. H_2O

After insufflation of 500 cc. ÷ 6 and + 1 cm. H_2O

Insufflation was not repeated.

Jan. 23. Feels well. *Ausc.:* Suppression of resp. only over upper part of right lung. *X-ray exam.:* Air of pneumothorax almost completely absorbed.

Febr. 13. *X-ray exam.:* Rather marked veiling of right apex and slight veiling of left apex; no sign of infiltration. Slight diffuse veiling of entire right lung field. Both hilus shadows markedly compact and containing areas of calcification. No sign of pneumothorax. *Expect.:* ÷ T. B. (13 times).

March 20. Discharged.

After-treatment in Boserup Sanatorium March 22— July 13, 1923.

Tp. normal. *Expect.:* ÷ T. B. *Ausc.:* (on discharge): Short perc. note over right apex; no other abnormality.

After-exam. May 30, 1928: Perfectly well during past 5¼ years. *Ausc.:* No abnormality. *X-ray exam.:* No definite sign of any pathologic process, recent or old. It may be that a mild degree of emphysema is present (Dr. med. *Panner*).

Summary: Acute onset of right-sided pneumothorax with moderate displacement. Adm. 1 week after onset. No exudate made out on auscultation, but X-ray plate shows shadow of minimal effusion. Afebrile course. Rapid absorption of air. After-exam.: Well during past 5¼ years. No sign of tuberculosis.

CASE 21.

Male, 36 years, blacksmith.

Københavns Amtssygehus, Frederiksberg, Med. Dep., Aug. 25, 1923—Oct. 2, 1923.

Father and 1 sister died of rapidly progressing pulmonary tuberculosis, 16 years ago. Past history of good health, apart from attacks of dyspepsia.

4 days ago, marked pain on resp. in right half of chest, lateral to nipple. As pains persisted, he consulted physician, who diagnosed pleurisy. Pt. did not feel sick otherwise, and has done his work during these days too.

On adm.: Looks natural. Tp. 37,5: P. 76. *Ausc.:* Left lung normal. On right ant. surf., resp. almost completely masked by strong friction sounds, especially around the nipple. Friction sounds on post. surf. too. No dullness. No râles. Heart findings normal.

Aug. 26. Pain only on deep resp.. *Ausc.:* Right ant. surf., friction sound and suppressed resp. from C.III to C.VI. Right post. surf. faint resp. from apex to base, with a little short note.

Tp. perfectly normal throughout stay in hosp.

Pirquet: +. *Expect.:* ÷ T. B. (3 times).

Sept. 7. No stitch. *Ausc.:* Short tone over right apex and a few fine moist râles in right IC.I. Faint resp. and lowered vocal fremitus on right ant. surf. from C.III and on post. surf. from scapular angle.

Sept. 8. *X-ray exam.:* Right lung field, lower portion quite clear, without trace of lung markings, upper portion bordered by a distinct line from C.IV (pneumothorax). Above this line, condensed lung field. Left lung field veiled, dense hilus shadows.

Sept. 21. *Ausc.:* Practically the same as on Sept. 7.

Oct. 2. Discharged.

Subsequent history: 5 months later, March 6, 1924, pt. adm. for bronchopneumonia to St. Josef's Hosp., Med. Dep. B, where he died on April 4, 1924.

Abstract of record: March 3: Sudden onset of illness with pains in loins, chill and signs of very severe pneumonia (influenza pneumonia?) of right lung, together with bronchitis of left lung. Tp. stayed around 39—40. Towards the last, additional signs of right-sided pyo-pneumothorax.

Autopsy: Pyo-pneumothorax of right side. Right lung is a small atelectatic flap. On section, no sign of pneumonia, but pus in the larger bronchi. *No macroscopic tuberculous changes. Hilus glands enlarged, and firm, without apparent caseation. Left lung emphysematous. No other abnormality of lungs.* Mesentery contains a few large calcified glands. *Aut. diagn.:* Right-sided pyo-pneumothorax; Purulent bronchitis, right; congestion of liver; calcification of mesenteric glands.

Summary: Acute onset of partial, lower, right-sided pneumothorax. Adm. 4 days later. At first, friction sounds. Afebrile course without effusion. After being well for some time, pt. had — half a year later — attack of severe acute right-sided pneumonia with fatal issue. Pneumonia complicated with pyo-pneumothorax, such as is occasionally observed in influenza pneumonia. Autopsy showed no macroscopic signs of tuberculosis in the lungs.

CASE 22.

Male, 23 years, messenger.

Bispebjerg Hosp. Dep. B, September 7, 1923—Sept. 30, 1923.

2 uncles are thought to have suffered from pulmonary tuberculosis; otherwise no tuberculosis in family. Past history of good health.

One month ago, onset of "muscular rheumatism" in form of pains in right shoulder and right arm. At the same time, pt. felt short of breath, and had pain back of sternum. These symptoms did not set in particularly suddenly. Dyspnea and stitch in shoulder aggravated by exercise and deep resp. Treated a whole month with aspirin; but, as severe dyspnea persisted, pt. submits to X-ray exam. which shows total right-sided spontaneous pneumothorax.

On adm.: Looks natural. Tp. 37.3. *Ausc.:* Over entire right lung, light tympanitic perc. note; resp. partly faint, partly inaudible.

Tp. perfectly normal throughout stay in hosp..

Sept. 13. *X-ray exam.:* Total right-sided pneumothorax. Only very small shadow of right lung, protruding as a flap a little under the middle of the medial border. Left lung is long, slightly veiled, especially in middle part; dense hilus shadow with spots of calcification. Heart shadow somewhat enlarged to left, perhaps from displacement.

Sept. 21. *Expect.:* ÷ T. B.

Sept. 30. Discharged.

Subsequent treatment in Boserup Sanatorium, Nov. 9, 1923—March 8, 1924.

Pneumothorax disappeared within a couple of months. Tp. normal. Expect.: ÷ T. B.

After-exam., June 8, 1928: Perfectly well during past 4¾ years. *Ausc.:* Tone perhaps a little short over right apex. Lung borders slightly distended. No other abnormalities. *X-ray exam.:* No sign of infiltrative processes. Emphysema undoubtedly present. (Dr. med. *Panner*).

Summary: Rather gradual development of right-sided pneumothorax without particular displacement. Admitted 1 month after. Air absorbed in ca. 3 months. After-exam.: Well during past 4¾ years. No sign of tuberculosis. Mild emphysema.

CASE 23.

Male, 20 years, student.

Kommunehosp., Dep. III, Nov. 11, 1923—Jan. 10, 1924.

No tuberculosis in family. Past history of good health.

3 days ago, in morning, pains in left side of chest and in back, especially on deep resp.. Felt badly, worse in afternoon, but stayed up all day. Worse yet in evening, with increasing pain, dyspnea and headache. Tp. rose to 39°. No chill, but sensation of fever. While on the bedpan, in evening, pt. collapsed, and his landlady who assisted him, thought he was dying. She summoned Dr. *Harbou Hoff,* who watched him several hours. The following days, the

condition was less serious. No cough or expect. some dyspnea and palpitation. A couple of vomitings.

On adm.: Pt. is pale, somewhat dyspneic. Tp. 38.7; P. 100; Resp. 28. *Ausc.:* Left ant. surf., tympanitic perc. note from clavicle to base. Resp. almost inaudible. No. râles. Traube's space free. Left post. surf., Flat dullness from middle of scapula to base; resp. inaudible; no vocal fremitus. Right lung normal. Heart lung-covered. Rx. Mixt. camphor..

Nov. 12. 38.6/38.1. P. 116. No complaints. *Ausc.:* Heart dullness cannot be made out on perc. Heart impulse felt only to right of sternum, most distinct in IC.V, a little inside right nipple line. Outer border of heart dullness at right nipple line. *Expect.:* ÷ T. B.

Nov. 13. 38.9/37.9. Pale with cyanotic tinge. *Explor. Puncture:* Evacuation of 10 cc. of dark, blood-coloured fluid, like venous blood. (No bacteria. No growth.)

Nov. 14. 38.6/37.8. *X-ray exam.:* At level of C.VI post., horizontal surface of fluid extending across entire left lung field, with intense uniform density of underlying portion. Above this line, considerable accumulation of air, with collapsed lung medially. Right lung field free. Marked scoliosis of dorsal spine. *Blood exam.:* Sahli 53 %; Erythr. 2.5 mill.; Index: 1; Leucocytes 6.700. Dry prep.: Slight polychromasia; no other abnormalities.

Nov. 15. 39.0/38.2. Slight epistaxis. No hemophilia. in family. He does not bleed long when injured.

Nov. 16. 38.5/37.5.

Nov. 17. 38.4/38.1. *Tp. slightly elevated next 3 days, thereafter normal.*

Nov. 24. *Ausc.:* About the same as before. Ictus palpable only to right of sternum.

Nov. 26. *Puncture* on left side; *evacuation of 400 cc. of bloody fluid,* whereafter no more could be aspirated. Pressure about 0. Insufflation of 400 cc. air. *No more aspirations performed, but a total of 5 air insufflations into pleural cavity.*

Dec. 3. Sahli 85 %; Erythr. 4.5 mill.

Dec. 28. *X-ray exam.:* Only a little fluid in left side.

Jan. 8. Sahli 121 %.

Jan. 10. Feels perfectly well. Discharged. Instructed to be careful the first year, take vacation the first 3 months, and submit to physical exam. every month during the first year.

After-exam., May 13, 1928: Perfectly well during past 4¼ years. *X-ray exam.:* Emphysema, very distinct lung markings. No signs of recent or old tuberculosis. (Dr. *Paul Jacoby).*

Summary: Acute onset left-sided hemo-pneumothorax with large hemorrhage. Collapse ca. 12 hours after onset of illness. Febrile course first days. On 18th day, aspiration of 400 cc. of blood. After-exam.: Perfectly well during past 4¼ years. No sign of tuberculosis.

CASE 24.

Female, 25 years, servant.

Medical Policlinic, Rigshospital, March 11, 1924.

Father has had pleurisy. Pt. is No. 3 of 7 children. 1 sister died of tuberculosis, 1 sister suffering from pulmonary tbc. Past history of good health, espec. no scrofula.

Present illness began 4 years ago with sudden violent stitch in right side of chest; next 8—10 days, stitch and dyspnea on resp. and exercise. Since, 2 similar attacks, 2 and 1 year ago. She states that she has been perfectly well between these attacks; she did not have fever nor keep her bed during previous attacks.

4 days ago, again stitch in right side of chest. The attack began with hiccough for ca. ½ hour, then pain in right lumbar reg., and on each resp. movement the pain extended higher and higher up in chest. The first days, continuous pains, whether at rest or at work. Pt. comes to Med. Policlinic for exam.

On adm.: Tp. 37.1. *Ausc.:* Resp. suppressed over entire

right side. *X-ray exam.:* Total right-sided pneumothorax. Heart slightly displaced to left. No effusion.

Pt. then treated in *Rigshospitalet, Med. Dep. B, March 15, 1924—May 6, 1924.*

March 19. Up. For a long time, pain in right side, most in back under scapula. Tp. normal, excepting a rise in evenings to 37.6—37.7 during week preceding menses.

April 2, *Ausc.:* Still tympanitic note on right post. surface, feeble resp..

April 8. *X-ray exam.:* Pneumothorax subsiding. No other abnormalities.

April 24. *X-ray exam.:* No pneumothorax. Lung markings distinct clear out to chest wall.

April 30. Pain in right infrascapularis on deep resp. *Ausc.:* No abnormality, especially no friction sounds.

May 6. Discharged.

Pt. was then perfectly well till spring, 1927, when she began to suffer from cough, fever and dyspnea. *Expect.:* + T. B.

Pt. then in *Øresundshospitalet, Med. Dep., June 4, 1927 —Nov. 28, 1927* for *left-sided pulmonary tuberculosis.* There were scattered infiltrations in left lung and a cavity (size of pigeon egg) at level of C.III. *No definite abnormality in right lung.* Tp. elevated. Sedimentation test: Increased values. *Expect.:* + T. B. Pt. in bed all the time while in hosp. Requested discharge, in spite of advice to stay.

Pt. then at home about 1 month, till adm. to

Kommunehosp., Dep. III, Jan. 5, 1928—June 10, 1928.

The record states that the 3' sister was suffering from pleurisy in summer 1927.

On adm.: Pt. had high tp. and was in bad shape from extensive laryngeal tuberculosis. *Ausc.* and *X-ray exam.* showed extensive progressive pulmonary tuberculosis with cavity formation of left lung, but no definite abnormality in right lung.

Jan. 26. *Artificial Pneumothorax* on left side with excellent effect on all symptoms.

Pt. discharged for after-treatment in Boserup Sanatorium.

Pt. treated in *Boserup Sanatorium from June 19, 1928, till her death, September 8, 1928.*

On adm.: X-ray exam. revealed no definite abnomalities in right lung; left lung showed artificial pneumothorax and in the upper lobe a large cavity, not completely compressed.

Pt. under usual sanatorium treatment; and in addition, insufflation of air was performed 3 times to maintain the artificial pneumothorax on the left side.

Sept. 6. Sudden rise of tp. to 40.8 in evening.

Sept. 7. Tp. stays between 39 and 40. Pains in various parts of body. Considerable shortness of breath; P. 132. Slight cyanosis.

Sept. 8. Very marked dyspnoea and a loud rattling in the throat. P. 120, fairly strong; Resp. 40. Troublesome expectoration. No particular pains. During the forenoon, pt. falls off markedly, and at noon the dyspnoea is aggravated. Death comes at 12.20, quiet and calm.

Autopsy. Sept. 10: Opening of the thorax reveals a pneumothorax on the right side. The entire lung is compressed, filling but half of the right pleural cavity. At the junction of the upper and middle thirds of this lung are some fine adhesions to the lateral chest wall. At the base of the lung, resting on the diaphragm and protruding out to the side in the air-filled space, there is a large and very thin-walled vesicle (about the size of a goose-egg), which is found to consist of distended lung tissue from the lower lobe, which is the site of emphysema. The middle lobe is studded with tuberculous foci, caseated and calcified, ranging in size up to that of a large pea.

There is no fluid in the pleural cavity. The site of the rupture in the lung can not be made out. The *left* side of the chest shows pneumothorax, with the lung occu-

pying about half of the space. The upper lobe is situated as a sausage suspended by the apex and adherent to the lateral chest wall through 3—4 strong adhesions. On section, this lobe is found to consist almost entirely of a thick-walled pus-filled cavity. The lower lobe shows numerous tuberculous foci. No fluid in the chest cavity. Both kidneys rather small in size, the other organs appear normal.

Autopsy diagnosis: Pneumothorax spont. dext. Pneumothorax artificialis sin. Tuberculosis pulm. dupl. cavernos. lobi sup. sin.. Emphysema pulm. lobi inf. dext.. Pleuritis adhæsiva duplex. Hypoplasia renis duplex.

Summary: Acute right-sided pneumothorax with slight displacement, probably 3 previous attacks in the past 4 years. No exudate. No sign of tuberculosis. Air disappeared in about 6 weeks. Then perfectly well for 3 years, till she had acute progressive cavernous phthisis of the left lung (pt. belonging to a markedly consumptive family). At this time, no demonstrable abnormality in right lung. ¾ year later, as pt. is steadily getting worse, artificial pneumothorax is instituted on left side, with good effect. After the artificial pneumothorax has been maintained more than 7 months, pt. had, in connection with an acute febrile condition, a sudden attack of acute spontaneous pneumothorax on right side, and she died. Autopsy findings: Left-sided artificial pneumothorax, occupying about half of the space. Upper lobe of left lung consisting almost entirely of a thick-walled, pus-filled cavity; lower lobe containing many tuberculous foci. In addition, right-sided spontaneous pneumothorax. Right lung compressed to half its normal size. No fluid in pleural cavity. Site of pleural rupture is not found. Numerous tuberculous foci in middle lobe of right lung. Right lower lobe shows emphysema; and here, at the base of right lung is a large, very thin-walled vesicle (about the size of a goose-egg), resting on the diaphragm and protruding laterally out in

the air-filled pleural cavity. This vesicle consists of distended lung tissue.

For discussion of this case, see p. 116.

CASE 25.

Male, 32 years, farmer.

Hornsyld Hosp., May 13, 1924—Aug. 13, 1924.

No tuberculosis in family. Past history of good health.

2 weeks ago, sudden onset of present illness. Pt. awoke at night with marked air hunger and pressure pain in left side of chest. Tp. was normal. The pressure pain persisted next 5—6 days, but pt. felt fairly well. One week ago, he was getting worse with increasing dyspnea and rise of tp. to 39,3. No cough or expect.. Condition improving slowly.

On adm.: Looks natural. Tp. 36,7; P. 80; Resp. 24.

Ausc.: Thorax of normal shape; excursions egal. Heart lung-covered. Tympanitic perc. and feeble resp. over entire left lung. No râles or friction sounds. Marked metallic tinkling over entire left side, no Hippocratic succussion splash.

May 15. *X-ray exam.:* Left lung completely collapsed, compressed against hilus; some protrusion of heart on right side. No sign of fluid.

Intrapleural pressure on left side: Average ÷ 3½ cm. H_2O.

Tp. perfectly normal throughout stay in hosp.

May 23. *X-ray exam.:* Freely movable exudate, reaching on ant. surf. to C. V. Lung less compressed. Heart not further displaced.

May 30. *Pneumothorax-Insufflation:* Initial pressure: ÷ 2½ cm. H_2O. After 350 cc. of air: ÷ ¼ cm. H_2O. Only this one insufflation performed.

June 23. Feels well. *Ausc.:* Some friction sounds over lower left ant. and post. surf. . Feeble resp. over lower post. surf.

Aug. 13. Feels well. Discharged.

After-exam. Nov. 14, 1928: Perfectly well during past 4¼ years. *Ausc.:* Normal findings. *X-ray exam.:* No abnormality.

Summary: Acute onset of left-sided pneumothorax with slight displacement. Adm. 2 weeks later. 3 weeks after onset of illness, X-ray exam. reveals an exudate that was not observed before. Tp. normal. After-exam.: Perfectly well during past 4¼ years. No sign of tuberculosis.

CASE 26.

Male, 22 years, shoemaker.

Skive Hosp., Med. Dep. Febr. 15, 1925—March 25, 1925.

A. No tuberculosis in family. Past history of good health.

On July 16, 1924, in afternoon, *sudden stitch* in right side of chest during work. Pt. had not exerted himself that day, but he had strained himself in play a couple of days before. At night, he still had some indefinite pains in right side of chest; he ate 2 beefsteaks and went to bed early. Later, at 10 o'clock, he had violent pains, localized in right side of epigastrium and entire right side of abdomen. He had very severe pains on the slightest motion, and on resp. He was adm. to hosp., where a diagnosis was made of perforation of gastric ulcer, and laparotomy was performed immediately after adm. It was ascertained that the pt.'s lesion consisted in a right-sided spontaneous pneumothorax; and puncture was made the following day. One month later, pt. had almost completely recovered. A couple of months later, he was taken soldier, dragoon, but proved very soon to be completely incapable of exertion connected with service; so he was discharged.

B. Febr. 15, 1925. *3 hours ago, sudden onset* of present attack, with severe pains in right side of chest, setting in immediately after lifting a heavy load. Was at once very pale and markedly dyspneic; dyspnea subsiding some-

what the next couple of hours. These symptoms were accompanied by a protracted chill and some cough. No expect.

On adm., 10 P. M.: Not particularly exhausted but quite pale, with cyanotic hue. Tp. 37,4; P. 112; Resp. 28. *Ausc.:* Resonant perc. note on right side, ant. to just above curvature, post. to C. XII.; feeble resp. . Lagging of right side on resp. No râles or friction sounds.

Febr. 16. Only slight dyspnea and cough. Evening tp. 38,3.

Febr. 17. Tp. 36,8. Slightly cyanotic, slightly dyspneic. *Ausc.:* Some displacement of heart to left.

X-ray exam.: Total right-sided pneumothorax with displacement of mediastinum and partial compression of left lung. *Tp. perfectly normal.*

Febr. 21. *Puncture.*
Some air escapes, but pt. gets pale and cyanotic under puncture so this is discontinued.

Febr. 28. *Ausc.:* Resp. audible only over apex and along right sternal border.

March 11. Up.

March 25. Feels well. Discharged.

Apart from evening, Febr. 16, *tp. perfectly normal throughout stay in hosp.*

Re-adm. July 14, 1926 for exam., by request of Invalid Relief Council: In Oct. 1925, a similar attack as before. Incapable of work on account of violent stitch in right side and dyspnea. *Ausc.:* Perhaps slight suppression of resp. over right lung; no other abnormality.

After-exam. Nov. 17, 1928: Since last, one more attack of same kind, but less severe. Is very nervous, and tires readily; incapable of work on account of pain in right half of chest and dyspnea. *Ausc.:* No abnormality, except perhaps slight suppression of resp. over right lung. *X-ray exam.:* No abnormality.

Summary: July 1924: Acute onset of right-sided tension

pneumothorax. Diagnosed as perforating gastric ulcer, wherefore laparotomy. 7 months later, recurrence of right-sided tension pneumothorax. Since then, pains in right side and dyspnea, incapacity for work. Probably 2 subsequent attacks. After-exam.: No definite abnormality. No sign of tuberculosis.

CASE 27.

Male, 31 years, shoemaker.

Frederiksberg Hosp., Med. Dep. B., July 22, 1924—Aug. 9, 1924.

A. No tuberculosis in family. Past history of good health. One year ago, adm. to this dept. for dyspepsia. At that time, normal findings on ausc.

2 days ago, sudden onset of pain in lower part of back, over loins and radiating to both sides, equally. Pains were not very severe and set in without preceding exertion. Pt. slept well the following night, but yesterday the pains were so severe that he had to quit work. Pains are less severe to-day, but are aggravated on motion, which also makes him short of breath. Slight cough and mucous expect., slightly blood-tinged on the first day.

On adm.: Looks somewhat emaciated. No cyanosis. Dyspnea on motion. Tp. 37,5. *Ausc.:* Tympanitic and faint amphoric resp. over entire right lung; ant. border C. VIII, post. C. XII. Left lung normal. Heart border C. III. midline. Ictus in IC. V in nipple line.

Tp. perfectly normal throughout stay in hosp.

Aug. 7. *X-ray plate (cf. Fig. 27):* Extensive pneumothorax of right side with complete compression of lung. Left side: No sign of infiltration, apart from a few calcified foci. Only very slight displacement of heart to left. *Expect.:* ÷ T. B. (2 times).

Aug. 9. Discharged.

Re-adm. for observation Dec. 13—Dec. 18, 1924. *X-ray*

exam.: No sign of pneumothorax of right side. Lungs suggestive of emphysema; no other abnormality.

B. *Re-adm. for spontaneous pneumothorax — this time on left side,* Jan. 19, 1925—Febr. 12, 1925.

Yesterday, at 5 P. M., *sudden onset* of pains in left side of chest, gradually extending over entire thorax, accompanied by marked dyspnea.

On adm.: Pt. is pale, perspiring, short of breath. Complains of pains „all over". Tp. 38; Resp. 36; P. 120, a little irregular. *Ausc.:* Distension of left half of chest, lagging markedly. Resonant perc. note over entire left lung. Resp. amphoric on ant. surf., inaudible on post. surf. Heart lung-covered. Fine moist râles over base of right lung.

Jan. 20. 38,8/38,4. Profuse sweating. Dyspnea even at rest. P. 120. *Ausc.:* Heart border is 5 cm. to right of midline. Coughs very little, expect. a few greyish lumps with occasional blood-tinged streaks.

Tp. slightly elevated a couple of days more, from Jan. 25 perfectly normal throughout stay in hosp.

Jan. 23. *X-ray plate (cf. Fig. 28):* Total pneumothorax of left side with complete compression of lung. Considerable displacement of heart and trachea to right.

Jan. 30. *Pirquet:* ÷.

Febr. 11. Up.

Febr. 12. Feels well. No change in pneumothorax. Discharged.

Re-adm. to this dept. for acute bronchitis, Dec. 1925.

X-ray exam.: No definite abnormality. *Expect.:* ÷ T. B. (4 times).

After-exam. May 7, 1928: Pt. well during past 3¼ years. *Ausc.:* No abnormality. *X-ray exam.:* No definite abnormality *(Dr. med. H. J. Panner).*

Summary: Acute onset of right-sided pneumothorax with slight displacement. Absorption of air. Half a year later, acute left-sided tension pneumothorax. Absorption

of air. After-exam.: Well during past 3¼ years, since last attack. No sign of tuberculosis.

CASE 28.

Male, 27 years, student.

Frederiksberg Hosp., Med. Dep. B, March 8, 1925—March 18, 1925.

No tuberculosis in family. Past history of good health.

Yesterday morning, pt. awoke with moderate stitch in left side of chest. Later in the day, pains increased, especially on motion, and pt. was somewhat short of breath. During the night, aggravation of pains and dyspnea. No cough or expect. No pain when lying quietly.

On adm.: Looks natural. No dyspnea. Tp. 37,6; P. 96; Resp. 18. Blood pressure: 110/65. *Ausc.:* Left side lagging. Tympanitic perc. and feeble resp. over entire left lung. Amphoric resp. over left ant. surf. . Right lung: Lower ant. border at C. VII; otherwise no abnormality. Ictus not palpable. Heart-sounds heard most distinctly in IC. IV, ca. 2 fingers to right of right sternal border.

Tp. perfectly normal throughout stay in hosp.

March 11. Up.

March 14. *X-ray exam.:* Total pneumothorax of left side. Lung completely compressed except for adhesion at apex. Perhaps slight displacement of heart to right. On right side, a few pea-sized calcified foci; no other abnormality.

March 18. Discharged.

After-exam. May 9, 1928: Perfectly well during past 3 years. *Ausc.:* Moderate emphysema. No other abnormality. *X-ray exam.:* Suggestion of emphysema. No infiltration or abnormal veiling (Dr. *V. Holm).*

Summary: Acute onset of left-sided pneumothorax without particular displacement. After-exam.: Well during

past 3 years. No sign of tuberculosis. Moderate emphysema.

CASE 29.

Female, 20 years.

Sønderborg Statshosp., Surg. Dep., Aug. 7, 1925—Nov. 4, 1925.

No tuberculosis in family. Past history of good health.

5 days ago, pt. took *suddenly* ill while clearing the table after meal, getting a sudden stitch in lower right side of back. Pains particularly pronounced on resp., but subsiding rapidly, to a large extent. Has stayed in bed since, but tried to-day to go to her physician, who lives close by. Arrived in doctor's office, pt. was cyanotic and markedly dyspneic.

On adm.: Moderate pains in back. Looks natural. No anemia or cyanosis. Tp. 37,8; P. 88. *Ausc.:* Tympanitic perc. over right post. and lateral surf. . Feeble resp. over right lung; in infraspinata accompanied by a loud whistling note. No râles. Left lung normal. Heart borders C. IV, left sternal border. Ictus outside nipple line.

Aug. 8. *X-ray plate* (cf. Fig. 20): Total right-sided pneumothorax with complete compression of lung. Considerable displacement of heart. No adherences. No exudate. No infiltrations in lungs.

A few evenings during first week, slight elevation of tp. (37,7|37,8); then tp. perfectly normal throughout stay in hosp.

Aug. 22. Lung expanding well. Up.

Oct. 13. *X-ray exam.:* Complete expansion of right lung.

Nov. 4. Discharged.

After-treatment in Ry Sanatorium, Dec. 3, 1925—Jan. 28, 1926.

Ausc.: Slight dullness over right apex; bronchial touch to resp. . No râles. No cough or expect. *Pirquet:* +. Sedi-

mentation test: 2 mm. *X-ray exam.:* No definite abnormality.

After-exam. Nov. 13, 1928: Perfectly well during past 3 years. *Ausc.:* Normal findings. *X-ray exam.:* No definite abnormality.

Summary: Acute onset of right-sided pneumothorax with rather marked displacement. Air absorbed in ca. 2 months. After-exam.: Well during past 3 years. No sign of tuberculosis.

CASE 30.

Male, 28 years, baker.

Randers Hosp. Octob. 3, 1925—Febr. 16, 1926.

No tuberculosis in family. Past history of good health. Has had some cold during the past week.

4 days ago, sudden stitch in right side of chest, setting in as pt. was turning on his side while resting on a couch. At the same time, marked shortness of breath. Dyspnea increasing next 2 days and persisting, preventing any work. Even when resting quietly, pt. feels unwell and has a continuous stitch in right side of chest. Some dry cough. No expect.

On adm.:, pt. is thin, slightly cyanotic. Tp. 38,1; P. 104/108; Resp. 32/34. *Ausc.:* Right side of chest slightly distended, immobile during resp. Slight dullness over apices. Over entire right ant. surf., in right axilla and infrascapularis, flat tympanitic perc. note. Feeble bronchial resp. in axilla and infrascapularis, with amphoric touch. No râles. No succussion splash. Ictus in IC. VI outside nipple line, marked. P_{II} slightly accentuated. Liver extends 4 fingers below curvature.

Oct. 4. 38,1/37,3. No dyspnea or cyanosis. P. 76.

Apart from first day, tp. perfectly normal throughout stay in hosp.

Oct. 7, *X-ray exam.:* Large right-sided pneumothorax.

No exudate. A snip of the compressed lung appears to be fixed, but no adhesion is distinctly demonstrable. Several spots and gland shadows at left hilus (tuberculosis?).

Oct. 23. Right lung begins to expand a little.

Nov. 3. *Ausc.:* Slight tympanitic note on right post. surf.

Dec. 21. *X-ray exam.:* Right lung fully expanded. Hilus shadows quite extensive. Lung tissue of both sides rather spotted and streaky. Veiling of left apex.

Dec. 26. Up.

Febr. 16. Feels well. Discharged.

After-treatment in Silkeborg Sanatorium, Febr. 19, 1926—April 16, 1926.

Ausc. on adm.: Slight dullness and feeble resp. over entire right lung. A single crepitus after cough under left clavicle. Tp. normal throughout. T. B. not demonstrated. *Ausc. on disch.:* The same as on adm., but no crepitation.

After-exam., Nov. 16, 1928: Perfectly well since discharge. *Ausc.:* Perfectly normal. *X-ray exam.:* As before, rather large hilus shadows. Some streaks in lower part of both fields. No other abnormalities.

Summary: Acute onset of right-sided tension pneumothorax. Some spots at hilus and streaks in lung tissue. Afebrile course without exudate. Air absorbed in 11 weeks. After-exam. Well during past 3 years. No sign of tuberculosis.

CASE 31.

Male, 47 years, labourer.

Lemvig County Hosp., Febr. 11, 1926—March 30, 1926.

No tuberculosis in family. Past history of good health. Some cold and cough the past week.

3 days ago, in morning, when pt. was getting up, he

felt a *violent stitch* around right scapula and noticed that he was getting short of breath. He has not kept his bed. Dyspnea somewhat subsided, but is aggravated by walking. And yet pt. walked all the way to hosp. (3 km).

On adm.: Pt. is large, robust, with cyanotic hue. Tp. 37,7; Resp. 24. *Ausc.:* Slight distension of right side of chest, borders: C. VII/C. XI. Resonant tympanitic perc. . Resp. amphoric, with whistling note. No râles. Left lung normal. Heart lung-covered. Ictus in IC. V, 2 fingers outside nipple line. *X-ray exam.:* Total pneumothorax of right side, pushing right lung and mediastinum towards left. Medial lung border along right border of vertebral column. Diaphragm not pushed down.

Febr. 12. 37,7/37,2. *One evening tp. 37,7, otherwise perfectly normal throughout stay in hosp.*

Ausc.: Perc. a little resonant over right lung, but not much. Resp. suppressed on right side.

Puncture on right side.

Intrapleural pressure is + 1 cm. H_2O, with a respir. variation of ½ cm. Aspiration of 150 cc. of air. After this, intrapleural pres. is still + 1 cm.

Febr. 22. *X-ray exam.:* Lower part of lung beginning to expand a little.

March 24. Up.

March 30. Discharged.

After-exam., Nov. 18, 1928: Perfectly well during past 2½ years, and has worked in gravel pit and on farm. *Ausc.:* Mild emphysema; no other abnormality. *X-ray exam.:* No abnormalities.

After-exam., Sept. 24, 1931: Perfectly well since last exam., without a single day of sickness. *Ausc.:* Slight emphysema; no other abnormality.

Summary: Acute onset of right-sided pneumothorax with moderate displacement. After-exam.: Well during past 5½ years. No sign of tuberculosis.

CASE 32.

Male, 19 years, electrician.

Københavns Amtssygehus, Frederiksberg, Med. Dep., March 9, 1926—May 12, 1926.

Mother's brother was suffering from tuberculosis while pt. was boarding with him. Pt. is the oldest of 9 children, of which 1 has scrofula, while the others are well. Till he was 10 years, pt. had rather large glands of the neck. When 4—5 years, pt. had whooping cough. Next year a physician noticed that *pt.'s heart was located in right side of chest;* and all the following years pt. has felt himself that his heart is in right side of chest. At age of 9 years, pt. is said to have had an attack of pleurisy on right side, and was staying in bed at home for 3 weeks. When 17 years, his physician said that he had pulmonary tuberculosis — without definite signs, however. Pt. was referred to sanatorium, but preferred to stay at home. Last couple of months pt. has suffered from attacks of dyspepsia and constipation, sometimes diarrhea. Adm. to hosp. under diagnosis of tuberculous peritonitis. No loss of weight.

On adm.: Rather marked functional dyspnea and sometimes feeling of skipping a heartbeat. No palpitation. Pt. is rather thin, but not poor. No dyspnea or cyanosis. Tp. 37.0; P. 84. Slight enlargement of a couple of cervical glands. *Ausc.:* Thorax asymmetrical, left half appearing larger than right half and more arched in upper part. Sinistroconvex scoliosis. Right lung borders extended: C.VII and C.XI. Over left lung, relatively clear perc., but impaired resp. from middle of infraspinata and from C.III to base. Impetus of heart seen on right side of sternum, visible and palpable ictus in right IC.IV about 2 fingers medially of right nipple, wherefrom dullness to C.III and right sternal border. Heart-sounds muffled, almost clear. No accentuations. No definite abnormality of abdomen.

March 11, *X-ray exam. (cf. Fig. 24):* Total pneumothorax of left side. Evidently marked intrapleural pressure, as entire left half of thorax is distended like a balloon; there

is a very marked bulging of mediastinum on right side, and the heart is completely pushed over in right side of chest.

Apart from a brief attack of angina, tp. was perfectly normal throughout stay in hosp. Pirquet: ÷.

Pt. was treated with dietary measures and — from March 24 — with light-baths. Pt. was on normal diet before discharge.

April 10. *X-ray exam.:* Conditions quite like before.

May 5. Tympanitic perc. and feeble resp. over entire left lung. No râles or friction sounds. May 12. Discharged. *Diagnosis:* Pneumothorax sin. Dislocatio cordis ad dext.. Scoliosis. Obstipatio.

After-exam., Jan. 6, 1929: Fairly well during past 2½ years. He was referred to tuberculosis sanatorium, but stayed at home, without working during first year. Apart from an occasional cold, he has been quite well. Last 1½ year he has worked as driver on delivery wagon. 1 year ago, he was already able to carry a load of 100 kg. from wagon into house. He carries the load on his back, and does this work right along. Gets short of breath only when trying to cycle fast. His pulse is mostly about 100. He is still rather thin.

Ausc.: Almost the same as 2¾ years ago, but left ant. and post. surf. somewhat flattened, almost immobile on resp. Feeble resp. over upper part of left side, inaudible over lower part. No râles or friction sounds. Clear perc. over left lung, ant. to C.VIII, post. to IC.XI. Right lung: Borders much distended, ant. to C.VIII, post to IC.XI. Slightly bronchial resp. in right supraspin.. No dullness. No râles or friction sounds. *Heart still in right side.* Heart dullness extends upwards to C.IV. Ictus 2 fingers medially of right nipple. *Lying down:* P. 84; Resp. 14. *Sitting:* P. 104; Resp. 16. *Standing:* P. 120; Resp. 18.

X-ray exam.: Left-sided pneumothorax involving entire left lung field. From apex to base, the clearance charac-

teristic of pneumothorax; but in lower half of the field also typical markings of blood vessels, suggesting that pneumothorax is only partial, and that there is also a probably crescent-shaped section of the lung. Considerable dislocation of heart as well as of mediastinum. (Dr. *V. Finsen*).

After-exam., Sept. 8, 1931: Apart from an accident with his motor cycle (in Aug. 1929), whereby he had a fracture of left clavicle, pt. has been perfectly well since last exam., gaining ca. 20 kg. in weight during this period.

In Sept., 1929, pt. felt a splashing in left side of chest; and this was so distinct that others could hear it. This splashing persisted about 2 weeks, giving no discomfort whatever. Ca. one year ago there was again a day when the splashing could be heard. Otherwise excellent health.

About ½ year ago, pt. noticed his pulse getting more slow, beating at a rate of 90—100, something he had never noticed before. On the whole he has been feeling fine, able to do any kind of work without having dyspnea. The only thing that bothers him is riding his bicycle against the wind. Has a cold now and then, but only for a day at a time. Outside of this, never any fever.

Pt. is robust and tall (181 cm.), very well-nourished.

Ausc.: Thorax somewhat barrel-shaped, without flattening. Resp. excursions almost symmetrical. No dullness. No râles or friction sounds. Lung borders: C.VII on both ant. surfaces; IC.XI on both sides post. Resp. sounds perfectly alike on both ant. surfaces. On back: Resp. sounds feeble in entire left infrascap., but no dullness; no râles or friction sounds; no succussio Hippocratis. No abnormalities over right post. surface.

Vital capacity: 3000 cc. Calculated normal vital capacity: 4525 cc. (height (in cm.) × 25).

Heart is lung-covered. Ictus not palpable. Heart sounds heard over normal place of heart. Heart sounds clear, regular. No accentuation. Pulse regular.

Lying down and sitting: P. 76—80; Resp. 12—14. Standing: P. 94; Resp. 12—14.

X-ray exam., Sept. 8 and 11, 1931: Pneumothorax, to the left of columna, in most post. part of pleural cavity; ca. 20 cm. in height (from diaphragm to C.IV on back), ca. 7 cm. in width, and about the same in ant.-post. diameter. So pneumothorax is nearly sausage-shaped; in both projections, lung markings are showing through, from ant. and lateral parts of lung. In bottom part of pneumothorax cavity (in standing position) is seen an exudate, ca. 3 cm. in height, which does not show on plates taken in lying position; in the latter plates the bottom of pneumothorax cavity is clear. Borders of pneumothorax cavity are fairly well defined and clear, with slight thickening of the wall. The surrounding lung shows nothing particular. Right lung appears perfectly normal. Heart shadow looks natural, showing only slight dextroposition (almost medial position). Upper part of mediastinum and trachea displaced a couple of cm. to the right, looking normal otherwise. (Dr. *E. Collin*). (*cf. Fig. 25 and Fig. 26*).

Summary: Chronic left-sided pneumothorax with displacement, developed at age of 5—6 years, discovered accidentally when pt. was 19 years. After-exam., 5½ years after discovery of pneumothorax, shows that there now remains only a partial left-sided pneumothorax. The lung has expanded partly, and it is functioning. No sign of tuberculosis. There is only a sausage-shaped remnant of the original pneumothorax, and this is situated up against columna, back of the heart, extending from diaphragm to C.IV. There is a small amount of fluid in this encapsulated pneumothorax cavity.

Pt. has been going about with a total left-sided pneumothorax for 17—18 years. Then, in the past couple of years, the lung has been expanding spontaneously, so that the remaining pneumothorax is only partial. No sign of tuberculosis.

CASE 33.

Male, 17 years, paperhanger.

Med. Policlinic, Rigshosp., March 22, 1926.

No tuberculosis in family. In 1925, radical operation for right otitis media. Jan. 1926, otitis on left side. Febr. 11, 1926, adm. to *Otolog. Dep., Rigshosp;* next day radical mastoid operation on left side.

Yesterday, sudden pain in left side of chest. Tp. 37,6. Referred to Med. Policlinic for exam. *Ausc.:* Slight tympanitic note on left side, especially on post. surf.. Resp. markedly suppressed over entire left side. Vocal fremitus lowered on left side. No Hippocratic succussion splash. Heart borders obliterated on left side. Heart-sounds heard over middle of sternum and to the right. *X-ray exam.:* Total left-sided pneumothorax. Small effusion shadow, in costodiaphragmatic angle. No abnormality in right lung.

First 4 days, insignificant rise of tp.; since tp. perfectly normal.

March 27. Up.

April 4. *X-ray exam.:* Marked decrease of pneumothorax.

March 18. Adm. to *Rigshosp., Med. Dep. A* for observation for kidney lesion. *Ausc.:* Perc. more resonant over entire left side than normal. Resp. feeble. *Pirquet* ÷. No sign of kidney lesion.

After-exam.: May 29, 1928: Perfectly well during past 2 years. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities (Dr. *V. Finsen*).

After-exam. Sept. 14, 1931: Perfectly well since last. *Ausc.:* Slight emphysema; otherwise normal findings. *X-ray exam.:* Moderate veiling of both apices and slight dimness of lung markings, suggestive of old fibrous changes; otherwise perfectly normal appearance of lungs; in particular, no sign of previous pneumothorax. (Dr. *E. Collin*).

Summary: Acute onset of left-sided pneumothorax without displacement. Air absorbed rapidly. After-exam.: Perfectly well during past 5½ years. No sign of tuberculosis.

CASE 34.

Male 34 years, blacksmith.

Øresunds Hosp., Med. Dep., May 28, 1926—Aug. 16, 1926.

No tuberculosis in family. Past history of good health.

Adm. physician states: *6 days ago,* at 8.30 A. M., pt. had *sudden onset* of pain in chest, dyspnea and cyanosis. Marked dullness and suppressed resp. over entire right lung. Tp. was normal; P. 140; Resp. 30. Rx.: Stimulants. May 23. 37,3/37,6. P. 100; Resp. 24. Less pain. No change in ausc.. May 24. 38,8/37,3. Resonant tympanitic perc. over right ant. surf.. May 25. 38,3/37,7. P. 96; Resp. 20. On right side, tympanitic perc. and suppressed resp. No râles. Liver dullness disappeared. Rise of evening tp. persists on following days. Pt. has been in bed all the time. There has been no cough or expect..

On adm.: Pt. well-nourished, not distressed, nor cyanotic. Tp. 37,9; P. 80; Resp. 20. *Ausc.:* Distension of right side of chest. For the rest, same findings as on May 25.

May 29. *X-ray exam.:* Right-sided pneumothorax, by which lung is compressed, so that it occupies only the medial part of the field below C.VII. No sign of adherences or exudate. Only slight displacement of heart to left.

Apart from adm. day, tp. perfectly normal throughout stay in hosp.

July 13. *X-ray exam.:* Lung has expanded a great deal. No definite sign of infiltrations in lungs.

July 28. *Ausc.:* Normal findings.

Aug. 5. *X-ray exam.:* No sign of pneumothorax.

Aug. 16. Discharged.

Subsequent history: Pt. perfectly well during first year after illness. No available information on later whereabouts of pt.

Summary: Acute right-sided tension pneumothorax. Air absorbed in little over 2 months. Well during first year after illness. Later whereabouts not known.

CASE 35.

Female, 22 years, clerk.
Frederiksberg Hosp., Dep. G. Jan. 5, 1927—March 10, 1927.

No tuberculosis in family. Pt. has congenital luxation of right hip. 12 years old, pt. had *left-sided exudative pleurisy.* She was in bed at home for 5 months; then in the *Christmas Stamp Sanatorium* at Juelsminde, Aug. 22—Oct. 14 1916. Here, slight dullness and feeble resp. in left infrascapula, but no other abnormal findings. Has been well since.

6 years ago, sudden onset of pains in right shoulder and marked dyspnea. Pt. was in bed 4 days, whereafter she was feeling well. During following years, several similar attacks of pain in right shoulder region, which the family physician takes to be rheumatism. On Dec. 3, 1926, adm. to Frederiksberg Hosp., Dep. F. for menstrual molimina. Pt.'s father asked the chief physician to examine her lungs in particular, with a view to these attacks. It was then ascertained that pt. was suffering from a right-sided coat-shaped pneumothorax. Dec. 22, 1926, *X-ray plate (cf. Fig. 13)* shows right-sided pneumothorax completely enveloping the lung. Lesion most pronounced in upper part of field, where apex is completely filled with air, and subsides downwards to base. Very small layer of fluid in left phrenicocostal angle. Heart completely displaced to left. In lower part of left lung, old calcification and streaky densities, probably due to fibrous changes. Calcification also present in apex and at hilus.

On adm. to Dep. G, Jan. 5, 1927: Tympanitic perc. over entire right lung with distinct metallic note. Ictus in IC.V, 3 cm. outside redressed nipple line. *Apart from premenstrual rise, tp. normal throughout stay in hosp.*

Jan. 12. *X-ray exam.: (cf. Fig. 14):* Very extensive pneumothorax of right side. Arch of diaphragm pressed down considerably, and entire right side markedly distended. No

further displacement of heart since last exam., but may be slight rotation.

Jan. 14. *Intrapleural pressure,* right side, about 0, with tendency to positive value.

Febr. 16. *X-ray exam. (cf. Fig. 15):* Very extensive pneumothorax of right side, which is inflated like a balloon. Lung very markedly compressed. Still, there seems to be a few fine adhesions of upper lobe. Marked displacement of heart and large vessels to left; suggestion of trachea being compressed at level of sternoclavicular joint. *Ausc.:* No changes.

March 8. *Ausc.:* Ictus in ant. axillary line. Discharged.

After-treatment in *Nakkebøllefjord Sanatorium.* March 10, 1927—April 22, 1927.

Ausc. and *X-ray exam.* showed quite the same conditions as found in Frederiksberg Hosp. General condition good. No cough or expect. Tp. normal. Sedimentation test: 1 mm. Vital capacity 1500 ccm. While in this Sanatorium, pt. had an attack of acute appendicitis and was adm. to *Faaborg Hosp.,* wherefrom she returned to her home.

Since then, pt. has come for regular control at *Frederiksberg Tuberculosis Station.*

July 23, 1927. *Ausc.:* Tympanitic perc. and feeble resp. over entire right lung. A few friction sounds over lower post. surf. *Ausc.* gave quite the same findings till Aug. 8, 1928. On next exam., Nov. 28, right lung had expanded.

After-exam. March 27, 1929: Perfectly well in past 2 years. *Ausc.:* Right half of chest somewhat retracted, with short perc. note. Resp. slightly impaired, vesicular. Questionable friction sound in right interscapular region. No râles. Left lung normal. *X-ray exam.:* Scattered calcifications in left lung. Fairly dense veiling of lower ¼ of both lung fields. Diaphragm more horizontal than usual and costodiaphragmatic angle obliterated. About centre of right lung field, a clear area with curved border, probably due to faulty projection, (Dr. *P. A: Blinkenberg*).

After-exam., Sept. 11, 1931: Perfectly well since last. *Ausc.:* No definite abnormalities. *X-ray exam.* (Sept. 11 and 29): Some diffuse thickening of right pleura; several calcified foci on left side; and some thickening of diaphragmatic pleura on both sides. Still, there can hardly be any exudate, as different oblique projections show the costo-diaphragmatic angle as being free; besides, the movements of the diaphragm are normal. No other abnormalities. (Dr. *E. Collin*).

Summary: At age of 12 years, left-sided exudative pleurisy. 10 years later, accidental notice of right-sided coat-formed pneumothorax. Probably several minor attacks during preceding 6 years. Extremely slow development, during 1 month, from coat-formed pneumothorax to extensive displacement pneumothorax. This pneumothorax persisted quite unchanged for 1¾ years, whereafter spontaneous expansion of the lung. After-exam.: Well during past 4½ years. No sign of tuberculosis.

CASE 36.

Male, 25 years, clerk.

Herning Hosp., January 12, 1927—Febr. 10, 1927.

Family history negative. Past history of good health, apart from kyphosis. Past 10 days, some cold and cough.

2 days ago, pt. awoke in night with very severe pain in left half of chest. Pain subsided very quickly, and pt. went again to sleep. Since then, pt. has suffered from increasing dyspnea, but kept working till last night, when he consulted physician, who adm. him at once.

On adm.: Slight cyanosis, marked dyspnea. Pt. of rather robust build. Tp. 38,7. *Ausc.:* Left half of chest distended. Resonant perc. over entire left side, distinct metallic note on perc.; resp. almost inaudible. Right lung normal. Heart displaced to right of sternum, where heart dullness is made out and heart sounds are heard distinctly, clear. *X-ray exam. (cf. Fig. 21):* Total left-sided pneumothorax. Lung

completely compressed, appearing only as a very faint shadow against lower part of mediastinum. Trachea markedly deviating to right. Heart completely pushed over in right half of chest, so that right heart border extends to 4 cm. from right chest wall. No sign of infiltration in right lung. Horizontal surf. of fluid in lower part of left pleural cavity at level of IC.V on ant. surf.

Shortly after adm. *Puncture.*

Escape of large amount of air, with a few squirts of blood.

After puncture, heart is still displaced to right of sternum; no change in perc.. Pt. feels greatly relieved.

Jan. 13. 38,7/38,5.

Jan. 14. 38,3/38. Feels well. No dyspnea. No cyanosis.

Jan. 15. 38,2/37,3.

Jan. 16. 37,9/36,9.

Jan. 17. *X-ray exam.: (cf. Fig. 22):* Heart almost returned to its normal position. Left lung is now appearing as a prominence — a little larger than a child's hand — to left of heart shadow. Tp. was about 38^0, a few evenings of the following week; and perfectly normal throughout rest of stay in hosp.

Jan. 27. Mean intrapleural pressure, 0 cm. H_2O.

Febr. 6. Sedimentation test: 1 mm.

Febr. 10. Discharged.

March 23. *X-ray exam.:* Left lung halfways expanded.

May 24. *X-ray exam.:* Left lung completely expanded.

After-exam.: Nov. 15, 1928: Perfectly well during past 1¾ years. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities.

After-exam., Sept. 25, 1931: Perfectly well since last. *Ausc.:* Normal findings.

Summary: Acute onset of left-sided tension pneumothorax with small hemorrhage. Critical condition. Puncture. Air absorbed in 2—3 months. After-exam.: Well during past 4½ years. No sign of tuberculosis.

CASE 37.

Female, 49 years, merchant's wife.

Private pt. of Dr. Martha Nørgaard, March 1927.

Whooping cough, diphtheria and typhoid fever in childhood and youth. In later years rheumatism and hypertension. Otherwise, past history of good health.

Present illness began on a motor trip, with *sudden onset* of a stitch in lower left side of chest. After staying in bed a couple of days, pt. sent for doctor. There was no fever, but a vague troubling sensation in left side of chest.

March 24. *X-ray exam.* (1 week after onset of illness): Coat-formed pneumothorax of left side. Outer edge of lung is 3 fingers from chest wall. No adhesions. No effusion. No infiltrations in lung tissue.

Pt. stayed in bed 3 more weeks at home; tp. was normal; and she felt well.

After-exam. Nov. 16, 1928: Apart from an occasional, brief, stitch in the back, pt. has been well during past 1½ year. After-exam. is kindly made by Dr. *C. Fasting-Hansen: Ausc.:* Some flattening of upper part of right side of chest. Otherwise normal findings. *X-ray exam.:* No abnormalities in the lungs.

After-exam., Oct. 22, 1931: Well since last exam. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities.

Summary: Acute onset of left-sided, coat-formed pneumothorax. Air absorbed in 3 weeks. After-exam.: Well during past 4½ years. No sign of tuberculosis.

CASE 38.

Male, 21 years, baker.

Herning Hosp., March 28, 1927—April 24, 1927.

A. At age of 11 years, left-sided exudative pleurisy, treated at home; after-treatment in *Christmas Stamp Sanatorium at Kolding,* where he stayed April 25—Aug. 15, 1916. Diagnosis: Sequelæ pleurit. sin.; Infiltratio pulm. sin.? Chief Phys., Dr. *Th. Oldenburg* has kindly informed that on adm. pt. showed diffuse dullness with râles and a

few friction sounds over ant. and post. surf. of left lung. *X-ray exam.:* Dense shadow over left lung field from C.I to C.III, general diffuse obscurity over rest of field. Right lung field normal. Tuberculin test: +. No expect.

Pt. apparently well on disch.

Pt. has since been perfectly well.

This morning, while at work — bending over a kneading-through — *sudden stitch* in right side of chest. Stitch is still persisting, but no other symptoms. No cough or dyspnea.

On adm., at 8 P. M.: pt. is pale, but not distressed. No cyanosis or dyspnea. Tp. 37,7. Heart-sounds clear. Right heart-border 2 fingers to left of left sternal border. Ictus in IC.V, 3 fingers outside the nipple line. *Ausc.:* Tympanitic perc. and feeble resp. over entire right ant. surf. to C.VII. Feeble resp. over entire right post. surf., with tympanitic perc. in infrascapular reg., where there are a few fine moist râles after cough. No other abnormalities. *X-ray exam.:* Right-sided pneumothorax; distance between lung surface and chest wall 2—3 cm. Very small amount of fluid in sinus.

March 29. 37,7/36,8. Condition unchanged.

March 30. Sedimentation test: 1 mm. *Pirquet:* +.

April 1. 37,4/36,8. Sedimentation test: 5 mm.

Tp. perfectly normal throughout stay in hosp.

April 5. Up.

April 22. Discharged. (Transferred to sanatorium).

After-treatment in *Silkeborg Sanatorium,* April 26, 1927 —Aug. 26, 1927.

According to communication from Chief Physician, Dr. *Begtrup-Hansen,* the lung had fully expanded within one month. Tp. normal. No expect. Sedimentation test normal. *Ausc.* (on discharge): Slight dullness over entire right side, and faint resp. over upper part. A single crackling after cough around right clavicle.

X-ray exam.: Slight veiling of right lung, hilus shadows enlarged. No definite active processes.

After-exam. Jan. 25, 1929: Perfectly well during past 1¾ years. *Ausc.:* Normal findings. *X-ray exam.:* Picture not altogether quiet. Veil over left side. There seems to be no infiltrative processes anywhere on either side. X-ray findings most probably suggestive of pleurisy, or perhaps rather of condition resulting from pleurisy. No sign of pneumothorax (Dr. *V. Finsen*).

B. *On Nov. 12, 1929, pt. was again taken suddenly ill* with pains in entire right side, without any particular occasional cause. There was no dyspnea. Tp. practically normal. He consulted at once his physician, who wanted to adm. him to hosp., but there was no room. A couple of days later, he consulted another physician, who did not think there was anything particular wrong with him, but referred him to Tuberculosis Station for exam. According to communication from Chief Physician, Dr. *Begtrup-Hansen,* pt. was suffering from right-sided pneumothorax. *X-ray exam.* (Nov. 18.): Coat-formed pneumothorax of right side. Lung border 1½ cm. from chest wall. No exudate. Dense hilus shadows suggestive of chronic previous tuberculosis, but no infiltrative processes in lung tissue. Pt. felt well, had worked for 3 days, and was allowed to keep working. One week later, he returned for *X-ray exam.* Pneumothorax had now disappeared. He is feeling perfectly well.

March 8, 1930. Pt. states that he is doing fine.

After-exam., Sept. 25, 1931: Perfectly well since last attack. *Ausc.:* Normal findings.

Summary: At age of 11 years, left-sided exudative pleurisy. At age of 21 years, acute onset of right-sided, coat-formed, pneumothorax. Air not absorbed till 2 months after. Then perfectly well for 2¾ years, when he has again an acute right-sided, coat-formed pneumothorax. Air absorbed in less than 2 weeks. After exam.: Apart from last attack, perfectly well during past 4½ years. No sign of tuberculosis.

CASE 39.

Male, 39 years. fireman.

Frederiksberg Hosp., Med. Dep. B, April 8, 1927—April 23, 1927.

No tuberculosis in family. Past history of good health, apart from hernia.

9 days ago, sudden onset of illness with chill, high fever and stitch in left side of chest. Been in bed at home, treated with hot compresses. Tp. has been normal the last days. There has been a little expect. of rubiginous character.

On adm.: Pt. is rather pale. Resp. 28; Tp. 37,6; P. 76.

Ausc.: Over entire left side, ant. and post. surf., tympanitic perc. and faint resp. No dullness. No râles or friction sounds. No succussion splash. Right lung normal. Heart dullness absent. Heart-sounds inaudible.

April 9. P. 64; Resp. 20. No complaint. No dyspnea or cyanosis. *Ausc.* like yesterday.

Tp. perfectly normal throughout stay in hosp.

April 12. *X-ray exam.:* Extensive pneumothorax of left side. Upper and lower part of lung are markedly compressed, while middle part is adherent to middle third of chest wall, so that the lung is filling middle third of chest cavity. There is a small exudate. On right side, accentuated lung markings on account of congestion, but only a very slight displacement of heart to right side. (On X-ray plate, the mentioned exudate occupies only the costodiaphragmatic angle (H. Kjærgaard)).

April 16. Feels perfectly well.

April 19. Up.

April 23. Discharged.

After-exam. May 15, 1928: Pt. has been perfectly well during the past year. *Ausc.:* Normal findings. *X-ray exam.:* Special plate of apices shows distinctly a diffuse veil over right side; immediately above clavicle, a spot — about 2 cm. in diameter — apparently consisting of a conglomerate of smaller irregular spots. Around this spot, streaky and

reticular structures. This area is doubtless indicative of a tuberculous process of an older date, chiefly of fibrous character. Apart from this, the lung fields show no sign of abnormal veilings or infiltrative processes (Dr. *V. Holm*).

By comparing this X-ray exam. with the first X-ray plate, taken April 12, 1927, the old plate shows distinctly the aforementioned 2-cm. spot in right apex. The lack of an apex plate at the first X-ray exam. is probably the reason why this spot is not mentioned in the description (H. Kjærgaard).

After-exam. April 17, 1929: Pt. is still perfectly well. *Ausc.:* Normal findings. *X-ray exam.:* Special plate of apex shows streaky and meshy structures on right side, indicating an old, fibrous phthisis. No other sign of abnormal veiling or infiltrative processes (Dr. *Worning*).

After-exam., Sept. 9, 1931: Perfectly well since last. *Ausc.:* Slight emphysema; otherwise normal findings. *X-ray exam.:* Slight veiling of entire right side from apex to C.IX on back, subsiding diffusely, suggesting thickening of pleura. Hilus regions and perihilar lung markings somewhat denser than entirely normal, most resembling old fibrous peribronchitis. Otherwise no abnormalities; especially, no trace of pneumothorax. Entire left side appears perfectly normal. (Dr. *E. Collin*).

Summary: Acute onset of left-sided pneumothorax with air spaces at apex and base. Middle part of lung adherent. Old fibrous scar in right apex. After-exam.: Perfectly well during past 4¼ years. No sign of tuberculosis.

CASE 40.

Female, 31 years, factory-worker.

Bispebjerg Hosp., Med. Dep. B, May 2, 1927—June 16, 1927.

In 1923, "abdominal inflammation". 7 parturitions, last one in July 1926. In summer 1926, adm. to Kommunehosp.,

Dep. III, for „pneumonia". In fall 1926, adm. to Bispebjerg Hosp., Dep. C, for „stitch in chest". Ausc. gave no abnormal findings. Diagnosis incerta. In Bispebjerg Hosp., Dep. B, Jan. 22—Febr. 14, 1927. Complaint of pains in left femur. *Ausc.:* Normal findings. Diagnosis: Observation. Now, May 2, 1927, adm. under diagnosis of broncho-pneumonia.

8 days ago, pt. was taken ill, with oppression of the chest, dyspnea, some cough and a little expect. Repeated vomiting. Pt. does not think that she has had any fever. After she has been in bed, the cough and dyspnea have now subsided somewhat.

On adm.: Pt. looks rather exhausted. No cyanosis, but respiratory motions of alæ nasi. Some deafness of right ear after otitis. Tp. 37,5. *Ausc.:* Short perc. note over right ant. surf., where resp. is feeble. No râles. On right post. surf., perhaps a slight impairment of resp. Ictus in IC.V, within nipple line. Heart-sounds clear.

May 3. *Ausc.:* Heart findings normal. Right ant. surf.: Short perc. note above clavicle; impaired resp. over entire ant. surf., without râles. Post. surf.: Short perc. note at base of left side, no other perc. changes; resp. seems faint over lower right side; in one place below angulus, bronchial resp.. A single moist râle on right side.

X-ray exam.: Very extensive pneumothorax of right side. On this side, the lobes of the lung appear slightly separated. Left lung seems normal.

Slight elevation of tp. (max. tp. 38,1) the 10 first days, then normal till June 7, when pt. had otitis.

May 5. Resp. practically completely suppressed over entire right lung. But no change in the perc. note.

May 16. Up.

June 15. *Ausc.:* As before, no audible resp. over entire right lung. No dullness.

After-treatment in *Boserup Sanatorium,* June 21, 1927 —Aug. 26, 1927. *Ausc.* and *X-ray exam.:* Right-sided pneu-

mothorax. Lung expanding gradually. While in sanatorium, symptoms of salpingitis.

Pt. was operated in Bispebjerg Hosp., Nov. 14, 1927, with removal of the right adnexa.

Febr. 18, 1929. Pt. adm. to Medical Policlinic, Rigshosp., when she again had signs of adnexa lesion. No chest symptoms.

After-exam. of the lungs, Febr. 27, 1929. *Ausc.:* Normal findings. *X-ray exam.:* No sign of pathological changes in the lungs (Dr. *K. Lehmann*).

After exam., Sept. 7, 1931: Except for a protracted attack of phlebitis, pt. has been well since last; in particular, there has been no symptom of any lung lesion. *Ausc.:* No abnormalities. *X-ray exam.:* No abnormalities. (Dr. *E. Collin*).

Summary: Acute onset of right-sided pneumothorax without displacement. After-exam.: No lung lesion during past 4¼ years. No sign of tuberculosis.

CASE 41.

Male, 29 years, bank clerk.

Odense Hosp., Med. Dep. B, May 2, 1927—May 15, 1927.

A. 2 cousins died of tuberculosis 4 years ago. Pt. had seen them only occasionally. No other case of tuberculosis in family. Past history of good health.

4 weeks ago, pt. took *suddenly ill* in the street while walking rather fast to the railway station. All of a sudden, he had violent pains in the chest. At first, the pains were not localized, but pt. subsequently felt them localizing to right side of chest. He had to stop at once to catch his breath, and he was at the point of fainting. He noticed that the resp. became almost twice as rapid as usual; but still, he did not feel too troubled to carry through the trip as planned, and he did not return till next morning. He had no fever or cough. On April 11, he consulted his physician, and he has stayed in bed at home for 3 weeks,

until now. He feels that he is incapable of deep resp., and of using his voice as normally. Occasional oppression of right side of chest.

On adm.: Looks natural, tp. 38; *Ausc.:* Right side of chest is markedly distended and takes practically no part in resp. Resonant perc. note over entire right ant. surf. from IC.II to 3 fingers below the nipple. Also resonant perc. extending in over sternum to one finger to left of left sternal border. Resp. much impaired. On post. surf., distension of right side of chest less conspicuous. Resonant perc. note down to IC.IX; Marked impairment of resp., except over lower part, where a whistling sound is heard. No dullness. Left lung normal. Liver not displaced.

May 4. *X-ray exam.:* Pneumothorax of right side with complete compression of right lung, apparently without adhesion and without fluid in pleural cavity. No displacement of trachea and heart.

Apart from admission day, tp. was perfectly normal throughout stay in hosp.

May 15. Discharged.

After-exam. (March 16, 1929): Well during past 1¾ years. 1 month after disch., pt. was examined by physician who found no sign of pneumothorax. *Ausc.:* Lung borders slightly extended on post. surf.; no other abnormalities. *X-ray exam.:* Horizontal ribs. Lung border at C.XI. Lung markings distinct. Long axis of heart somewhat more vertical than normal. All these findings suggestive of emphysema. Special plates of apices show a slightly diffuse veiling of right side, without any sign of infiltrative processes. No sign of pneumothorax. (Dr. *Worning*).

B. Pt. was then perfectly well till *May 26, 1929.*

In morning, while dressing, pt. had a sensation of „something coming loose" in right side of chest, with only slight pain. After being up about 1 hour, pt. went back to bed, as he realized that he had an attack of spontaneous pneumothorax. He was not particularly short of breath, but the pain increased during the day. Dr. *Svend Berner,* Ullerslev,

was called; he found definite signs of pneumothorax, but only slight tension, no displacement of heart, and no sign of pleural effusion. Tp. was normal. Pt. stayed in bed 1 month. *Tp. was perfectly normal throughout.*

July 1. Up.

Since then, pt. has always had a little pain on exercise, localized to right hypochondrium. No dyspnea or cough.

After-exam. July 16, 1929: *Ausc.:* Resp. a little feeble over entire right side; no other abnormalities. *X-ray exam.:* Pneumothorax over right apex and immediately below clavicle, limited by a lung shadow of upward and outward convexity. Obliteration of right costodiaphragmatic angle. Moderate emphysema. No sign of infiltrative processes (Dr. *V. Finsen*).

July 31. Feels well. *Ausc.:* Normal findings.

Sept. 6. Feels well. *Ausc.:* Normal findings.

C. Pt. was now perfectly well till *Oct. 29, 1930.*

On this day, *at 8,30 a. m.,* while eating his morning meal at home, he had suddenly an attack of diffuse pains in entire right half of chest, radiating out to right shoulder. He realized that this was a new attack of pneumothorax; so he went to bed at once. No dyspnea while lying quiet. *Physical exam.,* at 3,00 p. m.: Pt. lying on left side, to ease the pains. He is not in distress. Tp. 37,2; P. 80; Resp. 17. Diffuse distension of entire right side of chest, with tympanitic perc. to C.VIII and C.XII. Resp. inaudible. No dullness. No râles or friction sounds. No particular displacement of heart. Ictus indistinct in IC.V, just inside nipple line.

Oct. 30. Tp. 37,4/36,8; P. 68. Pt. slept well after Tabl. Cibalgini. Less pain. Very distinct metallic note of vocal sounds over entire right side.

Pt. stayed in bed the next 2 weeks.

Nov. 13. *Tp. perfectly normal throughout.*

Ausc.: No distension of right side. No sign of pleural effusion. Metallic note of vocal sounds persisting. Resp. heard more distinctly, still markedly impaired. Up.

Nov. 18. Feeling well, but slight dyspnea on walking.

Nov. 25. *Ausc.:* Resp. fairly distinct over most of ant. surface, and towards midline on back.

X-ray exam.: Right pneumothorax, lung collapsed to less than half its normal volume. No sign of adhesions. Lung markings show no abnormality, except a slight density at and around the hilus. No definite dislocation of mediastinum and trachea. Left lung shows a slight increase in density of hilus shadow, with fibrous streaks radiating upwards. (Dr. *Jens Nielsen*).

After-exam., Sept. 18, 1931: Perfectly well since last. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities. (Dr. *E. Collin*).

Summary: Acute onset of right-sided pneumothorax without displacement. Spontaneous absorption of air. 2 years. later, a new attack of acute right-sided pneumothorax without displacement. Spontaneous absorption of air. 1 year and 5 months later, a third attack of acute right-sided pneumothorax without displacement. After-exam.: Well during past 4½ years (apart from the two attacks of pneumothorax). No sign of tuberculosis.

CASE 42.

Male, 42 years, railroad man.

Kommunehosp., Dep. II, May 21, 1927—June 29, 1927.

A. No tuberculosis in family. Apart from scarlet fever, past history of good health.

2½ month ago, pt. had a bad cold. 1 month later, he consulted physician. At that time, he had some dyspnea when working and occasionally a stitch in right side of chest. Last 2 weeks, he has had severe attacks of very violent coughing with heaving and a couple of times with vomiting. Pt.can give no definite information as to whooping-cough, whether he has had this disease before, or with regard to an eventual source of infection. In connection with the coughing spells, he has expect. some "long white

slime". On the whole, most suspicious of pertussis. He went again to his physician, who adm. him under the diagnosis: Pneumothorax dextr.?

On adm.: Robust build. No cyanosis or dyspnea. Tp. 37,3; *Ausc.:* Heart findings normal. Lung borders distinct only on left side, where they seem extended a little. Perc. note perhaps a little more resonant on right side, at least in axilla. No dullness. Resp. almost completely inaudible on right side. No other abnormalities.

May 22. *X-ray exam.:* Very marked right-sided pneumothorax, loosening the lung from the lateral chest wall. The lung completely collapsed and appears as a prominence on mediastinum from hilus to diaphragm. No adherences. No exudate.

Tp. perfectly normal throughout stay in hosp.

June 15. *X-ray exam.:* No particular change since last exam. Absence of lung markings in right lung field, but marked displacement of mediastinum to left.

June 23. After *exsufflation* of 600 cc. of air, pressure is slightly negative. Total exsufflation of 700 cc. of air. Pressure is still slightly negative.

June 27. Marked decrease of dyspnea since exsufflation.

June 28. *X-ray exam.:* Lung expanded about half-way.

June 29. Discharged.

After-exam. June 8, 1928: Apart from pharyngitis, pt. has been perfectly well. *Ausc.:* Normal findings. *X-ray exam.:* Slight diffuse veiling of entire right side, but hardly sign of infiltrative processes, neither old nor recent .There appears to be sign of emphysema (Dr. *V. Holm*).

B. *Øresundshosp., Med. Dep.* Aug. 26, 1929—Sept. 16, 1929.

Pt. adm. for right-sided pneumothorax. Pt. perfectly well during past 2 years since preceding attack.

3 days ago, while lifting a heavy box, pt. had an attack of marked dyspnea and oppression of the chest and throat. Has not taken to bed. Went to doctor to-day. Complains of considerable dyspnea on exercise and oppression of right

side of chest. No palpitation. No cough or expect. No fever.

On adm.: Looks natural. Some dyspnea on motion in bed. Tp. 37,2; P. 56. *Ausc.:* Some displacement of heart to left. Tympanitic perc. over entire right lung. Feeble resp. over right ant. surf. to C.VI, on post. surf. to C.XI. No râles or friction sounds. Left lung normal.

Aug. 27. *X-ray exam.:* Extensive right-sided pneumothorax, apparently compressing the entire lower part of lung, so that the lung hardly occupies the medial half of pleural cavity below C.VIII. No adhesions made out. No exudate. No sign of pleural lesion. Heart is displaced to left.

Tp. perfectly normal throughout stay in hosp.

Sedimentation test: 3 mm. Expect.: ÷ T. B. *Pirquet:* +.

Aug. 30. Still some oppression of throat, but no other discomfort. *Puncture* in IC.IX, angular line. Pressure is + 4 and + 10. After *evacuation* of 900 cc. of air, pressure is ÷ 6 and + 1; but within a few minutes it varies around 0. Then insufflation of 600 cc. of ether-perfumed air, without being followed by odour of ether in expiration air. Next exsufflation of air to negative pressure; but like before, within a couple of minutes the pressure swings around 0.

Aug. 31. *X-ray exam.:* Right lung protrudes as a free arch in middle of right pleural cavity; at least a part of the lung is not so compressed as 4 days ago. No exudate. Left lung normal.

Sept. 4. Feels well. No oppression of the throat.

Sept. 12. *X-ray exam.:* Lung more expanded.

Sept. 16. Feels perfectly well. Discharged on request.

Sept. 30. *X-ray exam.:* Air of right-sided pneumothorax is absorbed. No particular pathological condition on either side.

C. *Kommunehosp. Dep. II,* Aug. 28, 1931—Sept. 4, 1931.

Pt. adm. again for right-sided pneumothorax. Perfectly well since last attack, 2 years ago. A few days ago, pt. exerted himself by moving a piano. Since then, he has been

dyspneic when walking fast or riding his bicycle. For this, he consulted his physician, who ascertained that he had again an attack of pneumothorax. Coughing only very little. No expectoration. No fever.

Physical exam.: Well-nourished. No cyanosis or distress. *Ausc.:* Left lung normal. Right lung: Strong tympanitic tone over entire ant. and post. surfaces and in axilla; no dullness; resp. inaudible. Heart: Apparently normal borders; action regular, 80. Heart sounds clear. Abdomen: Liver not below the curvature. No other abnormalities.

Tp. and pulse are perfectly normal throughout his stay in hosp.

Aug. 28. *X-ray exam.:* Right-sided total pneumothorax. No displacement of heart. On the screen, the right lung — completely collapsed — shows absolutely no changes from resp. phases.

Aug. 29. Coughing a little. No expectoration. Dyspnea on motion. Slight distension of right side of chest, ant. and post. Resp. excursions egal. To-day, no definite increase in tympanitic note over right side. Resp. greatly impaired, amphoric over one area.

Sept. 1. Sensation of oppression in right side of chest.

Sept. 2. *Exsufflation of 800 cc. of air from pneumothorax.*

Sept. 3. Greatly relieved, feels much better. *Ausc.:* Still considerable pneumothorax, tympanism and impairment of resp.

Sept. 4. No more discomfort. *Discharged.*

Sept. 4. *X-ray exam.:* Right lung expanded to about half its normal size. In the lowest part of the right lung field is a sharply defined ovoid contour, the interpretation of which is puzzling. (Dr. *T. Meyer*).

I am greatly obliged to *Chief Physician G. Biering,* Roentgen Clinic of the Kommunehospital, for calling my

attention to these very interesting pictures and for his kind assistance in their interpretation.

On further examination of these plates. in comparison with the earlier plates from the attack in 1927, my findings are as follows:

Even in the pictures from 1927, at the lower lateral margin of the lower right lobe one can make out the faint outlines of a bulging from the lung surface, about the size of a walnut.

On the last plates from the attack in 1931, one finds at the same site a configuration, about the form and size of a hen's egg (*see Fig. 11*). This body, protruding laterally from the lowest part of the lung margin out in the pneumothorax cavity, may hardly be interpreted as anything but a large subpleural vesicle situated on the margin of the lung. The shadow of the wall of this vesicle is so conspicuous that it must be fairly thick, fibrous in character, and probably containing some deposits of lime salts. The contour of this body is broken only in a small upper and lateral section. It is therefore reasonable to assume that in one area the otherwise thick wall of this large vesicle must have been rather thin, so that it does not show on the plate, and that the rupture of the vesicle has taken place in this thin area.

In all probability, then, one is really able to see on these X-ray plates the valve vesicle which by rupturing has given rise to the 3 attacks of spontaneous pneumothorax in this patient. On the plates from 1927, this vesicle can be seen as the faint outline of a walnut-sized configuration. During the intervening four years, till 1931, when the last attack of pneumothorax sets in, the vesicle has grown from the size of a walnut to that of a hen's egg, and at the same time the greatest part of its wall has been undergoing fibrous changes, so that it now gives a very distinct shadow on the X-ray plates. This roentgenographic finding is quite unique.

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Summary: Right-sided pneumothorax with displacement, developed during cold with coughing spells. Adm. 1—2 months later. Aspiration 3 weeks after adm. Well during next 2 years. Then again attack of right-sided pneumothorax with displacement, setting in after exertion. Aspiration 1 week later. Air absorbed in 1 month. Well during next 2 years. Then a third attack of right-sided pneumothorax, this time without displacement. Aspiration treatment. X-ray plates from 3' attack show a sharply defined configuration (form and size of a hen's egg) protruding from the lower border of the lower right lobe. This body must be taken as the valve vesicle which has given rise to the three attacks of pneumothorax in this patient. The vesicle is thick-walled, with a small thin-walled area. On the plates from the first attack, 4 years before, the vesicle is outlined but faintly, and only as large as a walnut. Apart from these attacks of pneumothorax, pt. has been perfectly well during the past 4¼ years. No sign of tuberculosis.

CASE 43.

Male, 40 years, chauffeur.

Usserød Hosp., Nov. 25, 1927—Dec. 19, 1927.

Father and mother died of tuberculosis. 2 other children living and well. Past history of good health.

2 weeks ago, while walking quite slowly, pt. had sudden attack of stinging pains in right side of chest. Pains were radiating up towards left shoulder, and out under left scapula. At same time, pt. noticed that he got short of breath on exertion. He kept working all day in spite of dyspnea, but pains were getting worse the following evening and night. After staying in bed a couple of days, stitch disappeared; but some dyspnea persisted. There has been no fever. No cough or expect.

On adm.: Robust build. *Ausc.:* Very slight dullness over entire right lung from apex to base, *not* increasing downwards. Resp. markedly impaired, almost inaudible, over

entire ant. surf. and in supra- and infraspinata. In right infrascapularis, on the other hand, resp. is distinctly bronchial with amphoric note, but still faint. Vocal fremitus somewhat lowered on right side. Normal findings on left side. *X-ray exam.:* Total right-sided pneumothorax without exudate; no other abnormalities.

Tp. perfectly normal throughout stay in hosp.

Dec. 19. Discharged.

Dec. 29. Physical exam. in *Rigshosp., Med. Policlinic. Ausc.:* Faint resp. over entire right side, which is lagging. Lung border on right side at C.VII and C.XI. Otherwise normal findings. *X-ray exam.:* Remnants of pneumothorax occupying right costodiaphragmatic angle. No definite sign of infiltrative processes anywhere, but there seems to be some streaky and perhaps maculated marking over apex.

Jan. 25. *Ausc.:* Normal findings. *X-ray exam.:* Pneumothorax apparently decreasing. *Pirquet:* +.

After-exam. April 29, 1929: Perfectly well during past year. *Ausc.:* Normal findings. *X-ray exam.* of apices and lungs shows no definite sign of pneumothorax or results from such. Lung markings, on the other hand, are extremely accentuated everywhere, and right apex shows several vague shadow formations, which are present in all the plates of this region. They are thought to be due to infiltrative processes, most likely of an older date (Dr. *Blinkenberg*).

After-exam., Sept. 12, 1931: Apart from a carbon monoxide poisoning, pt. has been perfectly well since last. *Ausc.:* Normal findings. *X-ray exam.:* No definite abnormalities, especially no sign of pneumothorax. Lung markings somewhat coarse throughout, in particular the bronchi at right side of hilus down toward the base, suggestive of fibrous changes. (Dr. *E. Collin*).

Summary: Acute onset of right-sided pneumothorax without displacement. Small fibrous scar in right apex. Air absorbed in 2 months. After-exam.: Well during past 3¾ years. No sign of tuberculosis.

CASE 44.

Female, 45 years, mail-carrier's wife.

Kommunehosp., Dep. III, Dec. 12, 1927—Dec. 24, 1927. and Jan. 6, 28—Jan. 13, 28.

No tuberculosis in family. Diphtheria and scarlet fever in childhood; otherwise past history of good health.

10 days ago, stitch in right side. 2 days later, pt. felt short of breath when she awoke in morning. During past week, pt. has suffered from shortness of breath and stitch in the back when ascending a stairway or walking in the street; still, she was able to do her work at home. She had no fever. Pt. had quite a similar attack with dyspnea 2 years ago, but recovered in 2 weeks without treatment. A couple of months ago, she had a similar, but brief attack.

On adm.: Pt. is well nourished, perhaps a little pale. Tp. 37,4; P. 76. *Ausc.:* Feeble resp. over right post. surf. from scapular spine to base, perhaps also over right ant. surf. Clear perc. over entire side. Heart border: Left sternal border, C.V, ictus in IC.VI, ca. 2 cm. outside midclavicular line. Heart sounds clear. No other abnormalities.

Dec. 8: *Ausc.:* Right side of chest is slightly distended; tympanitic perc. and faint resp. over entire side. No râles or friction sounds. Left lung normal.

Dec. 9. After cough, metallic note over and under right clavicle.

Dec. 12. *X-ray exam.:* Total pneumothorax of right side, with compression of entire lung. Heart is slightly displaced to left. No abnormal shadows in left side. No sign of infiltrative processes in the lungs.

Jan. 10. Faint resp. over entire right lung. Crackling râles after cough heard ant. over clavicle and post. in supra- and infraspinate reg. *X-ray exam.:* Pneumothorax is now occupying only upper portion of right pleural cavity. No other pathological processes observed in the lung.

Febr. 6. *X-ray exam.:* Right lung completely expanded. No sign of infiltration.

After-exam. Sept. 3, 1929: In Dec. 28, pt. had sud-

denly a stitch in right side, and some dyspnea. This was probably a mild relapse; it disappeared without pt. going to bed. Pt. has otherwise been perfectly well. Never fever, nor cough or expect. *Ausc.:* Occasionally an inconstant crackling is heard over both apices. Otherwise normal findings. *X-ray exam.:* No definite sign of infiltrative processes or abnormal veiling. Left half of chest, on the other hand, somewhat flattened, undoubtedly indicating some retraction. (Dr. *Kettel*).

After-exam., Sept. 3, 1931: Pt. thinks she has had one very slight attack since last exam. But she did not keep her bed. Otherwise perfectly well. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities. (Dr. *E. Collin*).

Summary: Acute onset of right-sided pneumothorax, with slight displacement. Air absorbed in little less than 2 months. Probably 2 previous and also subsequent slight attacks of pneumothorax. After-exam.: Well during past 3¾ years. No sign of tuberculosis.

CASE 45.

Male, 18 years, waiter.

Rigshosp., Med. Policlinic. Jan. 11, 1928.

Grandfather died of tuberculosis when pt. was 1 year old. No other case of tuberculosis in family. Pt. has always been delicate, and has been in the country twice for recreation. He has not been scrofolous. In 1923, exam. for pulmonary tuberculosis, and X-ray exam.: but no abnormal condition was made out. In April 1925, exam. in Rigshosp. Med. Policlinic, when he complained of pains in left side of chest. Some cough at that time, but no expect. *Ausc.:* Dullness over right apex and bronchial resp. No râles. Lung borders extended. *X-ray exam.:* No definite abnormality. Slight veiling of left apex, but no pathological significance attributed hereto. In June 1927, exam. again, on account of persisting cold. *Ausc.:* No abnormality. *Laryngoscopy:* Laryngitis, suspicious of tuberculosis.

Pt. returns to Med. Policlinic Jan. 11, 1928: Has been feeling well since last exam., coughing but little.

Day before yesterday: While waiting on a customer, pt. had a *sudden attack* of most violent pains in back and chest. He was not dyspneic. Went home and stayed in bed; comes now for exam. Pt. looks somewhat emaciated. Tp. 37,4. *Ausc.:* Heart is lungcovered. P_{II} accentuated. Systolic murmer at base. Lung borders ant. at C.VII, post. at C.XI. Resp. somewhat fainter over left post. surf. than over right. Otherwise no abnormality. *X-ray plate (cf. Fig. 17):* Upper portion of thorax looks somewhat broad. Ribs in horizontal position. Diaphragm on both sides at level of C.XI, and lung markings rather accentuated, as one finds them in emphysema. Heart shadow rather vertical, but without any other definite abnormality, perhaps slightly displaced to right. In left half of chest, lung markings are not observed in several places in the upper portion, towards the chest wall. These clear areas measure as much as 3—4 cm. in diameter; they occupy the entire apex and are defined against the normal lung tissue by blurred linear borders, and in some places by distinct shadow streaks. The case is undoubtedly one of spontaneous pneumothorax, which occupies the upper lateral portion of left pleural cavity, perhaps extending all the way down to diaphragm, as clear areas are met with all along the chest wall. It is difficult, however, to give any interpretation of the finding in the lower portion. No sign of infiltrative processes in the lung fields.

Feb. 6. Pt. returns after having stayed in bed at home for about 3 weeks. He has had no fever or expect., but coughs a good deal. *Laryngosc.:* Vocal cords of light rosa hue; barrel-shaped swelling of right vocal cord. Free motions. No infiltrations or ulcerations. No definite signs of laryngeal tuberculosis, but suspicion of beginning laryngeal tbc. *Pirquet:* +. Tp. normal.

Feb. 10. *X-ray exam.:* Pneumothorax now completely disappeared. Normal lung markings over the field. But,

about midways on left dome of diaphragm is a peculiar, conical, contour that is taken to be a pleural adhesion pulling the diaphragm upwards. This was not observed on previous examinations.

Feb. 17. Feeling well, apart from some tiredness.

Treated in Boserup Sanatorium, March 23, 1928—July 13, 1928.

Feeling well. Tp. normal. No expect. *Ausc.:* Short perc. note and faint bronchial resp. over both apices; no other abnormalities.

April 2. *X-ray exam.:* Slight veiling of right apex and enlargement of right hilus shadow with a few intense spots; nothing abnormal in the lung field itself. On left side, a few suspicious spots in apex. Left hilus shadow enlarged, with several intense extensions out into the field. On left side, diaphragm is indistinct and pointed in the middle. No sign of pneumothorax. Laryngosc.: Slight congestion of both vocal cords; no other abnormalities.

After-exam. Oct. 1, 1928: Started to work in September, but had to quit on account of pain in head and chest. Pt. gives an impression of neurasthenia. *Ausc.:* Heart is lung-covered. No other abnormalities.

After-exam. Febr. 8, 1929: Rather severe attack of influenza in January. *Ausc.:* Same findings as on Oct. 1, 1928.

After-exam., Sept. 21, 1931: Perfectly well since last. *Ausc.:* Moderate emphysema of the lungs. Lung borders: C.VII (ant.) and C.XI (post.). Heart is lung-covered. Otherwise no particular abnormalities. *X-ray exam.:* Well-defined densities in both apices, most marked on right side, as evidence of old infiltrations that have undergone fibrosis. On top of left diaphragmatic dome is a small funnel-shaped density, indicating the pull of a pleural adhesion. Slight enlargement of both hilus regions, most marked on left side, dense with swollen lymph glands. (Dr. *E. Collin* and Dr. *Worning*).

Summary: In adolescence, pt. was several times suspected

of pulmonary tuberculosis, but no such lesion was ever demonstrable. Acute onset of left-sided partial pneumothorax. Air absorbed in 3 weeks. After-exam.: Well during past 3½ years. X-ray exam. shows signs of old fibrous changes. Otherwise no sign of tuberculosis.

CASE 46.

Male, 18 years, student.

Private pt. of Dr. Svend Nandrup, Copenhagen, Febr. 13, 1928.

A. Parents and other children well. Father's sister died of pulmonary tuberculosis, but pt. did not come in close contact with her during her illness. Past history of good health.

Febr. 13, 1928. Sudden attack of stinging pains in precordium and at left curvature. Pains were at first moderate, but increased rapidly in intensity, so he soon had to go to bed. At that time, marked palpitation and some dyspnea. The physician first summoned took the case to be a heart lesion, and prescribed digitalis. That evening, pt. had already improved, and he slept well during the night. Was then examined by Dr. Svend Nandrup, who found feeble resp. over left ant. surf. with faint metallic ring. No râles. Pt. stayed in bed 1 week, was gradually getting less dyspneic. Tp. was normal all the time.

Febr. 20. *X-ray exam.: (cf. Fig. 16):* No sign of infiltrative processes. It is peculiar that in left apex as well as below clavicle, about down to the scapular angle, the lung markings are indistinct in the medial part of the lung, extending from hilus, and that the markings are completely absent in the lateral part and in the uppermost portion of apex. Apex plate shows a curved shadow formation just over the clavicle; this shadow runs across the lung field, is 3—4 mm. wide, sharply defined upwards, less distinct downwards, of upward convexity; absolutely no lung marking above this shadow. I can not imagine

anything else, but that these findings originate from a spontaneous pneumothorax, which has loosened the apex from the chest wall, and that the arched shadow is just the border of apex. Further down, however, the pneumothorax is localized so that it shows no distinct boundaries of the compressed lung. No abnormality observed in the lower half of the lung, especially no sign of pleurisy. (Dr. med. *H. J. Panner*).

Pt. stayed in bed 1 week more, with normal tp., whereafter he resumed his studies.

After-exam., Sept. 14, 1928: Perfectly well since. *Ausc.:* Normal findings. *X-ray exam.:* Lung borders somewhat extended. Heart perhaps somewhat vertical, suggestive of emphysema. No veilings or infiltrations in lung field. Hilus natural (Dr. *V. Holm*).

B. *Sept. 15, 1928,* (day after last exam.), while cycling slowly, pt. had a *sudden* attack of violent stitch in left side of chest, a little above curvature. With some difficulty, he managed to cycle home. I was summoned at once, and found the pt. slightly dyspneic, with superficial and painful resp. No cyanosis. Tp. normal. *Ausc.:* Slightly tympanitic perc. and feeble resp. over left ant. surf. from apex to C.III.

Next day, the subjective symptoms had almost disappeared.

4 days later, *ausc.* gave perfectly normal findings.

Pt. stayed in bed 2 weeks, with normal tp., feeling perfectly well; then convalescence for 1 week.

After-exam., Dec. 5, 1930: Perfectly well since last exam. (more than 2 years ago). Pt. has done military service for 19 months during this period, without any trouble whatever. *Ausc.:* Normal findings. *X-ray exam.:* No definite abnormalities.

After-exam., Sept. 7, 1931: Still perfectly well. Pt. is now non-commissioned officer in the army. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities.

Summary: Acute onset of left-sided partial pneumotho-

rax. Air almost completely absorbed 1 week later. 7 months later, a new attack of left-sided partial pneumothorax, which is absorbed very quickly. After-exam.: Perfectly well during past 3½ years. No sign of tuberculosis.

CASE 47.

Male, 30 years, civil engineer.

Private pt. of Dr. Oluf Gad, Copenhagen, March 30, 1928.

No tuberculosis in family. Past history of good health.

In Febr. 1919, pt. had a sudden attack of severe stitch in right side of chest, which subsided in a couple of days without any treatment.

March 30, 1928. While walking upstairs at home, pt. had a *sudden attack* of violent pains in right half of chest, localized just above curvature. At the same time, pt. had dyspnea and a little dry cough. On exam. 1 hour later, his physician found pronounced signs of right-sided pneumothorax, with resonant perc. over entire right ant. surf. and complete suppression of resp. No sign of effusion. Tp. 37,6. No expect.

March 31. *X-ray exam. (cf. Fig. 18):* Total right-sided pneumothorax without apparent adherences between the compressed lung and the chest wall. No abnormality made out in the compressed lung; no exudate in pleural cavity. Left lung normal.

The following evenings, slight rise of tp. (max. 38,3), whereafter normal tp. Some pain in right side of chest persisting the first days. *Pirquet:* ÷. *Sedimentation test:* Normal.

April 12. Up.

April 15. *X-ray exam. (cf. Fig. 19):* Considerable expansion of right lung, especially its middle „half". Lung markings more distinct. Nothing suspicious of tuberculosis. No apparent adhesions. No pleural effusion.

Later, pt. was exam. twice by Chief Physician, Dr. med.

G. E. Permin, who ascertained that the lung was gradually expanding completely.

After-exam., April 16, 1929:Perfectly well since.

Ausc.: Normal findings. *X-ray exam.:* No definite signs of pathological changes, especially no sign of pneumothorax (Dr. *V. Finsen*).

After-exam., Sept. 23, 1931: Perfectly well since last. *Ausc.:* Normal findings. *X-ray exam.:* No abnormalities apart from a moderate veiling of both apices, suggestive of old fibrous changes. There appears to be a little emphysema. (Dr. *E. Collin*).

Summary: Acute onset of right-sided pneumothorax without displacement. Lung expanding rapidly. After-exam.: Well during past 3½ years. No sign of tuberculosis.

CASE 48.

Female, 5 years, daughter of steward.

Svendborg Hosp., April 24, 1928—May 26, 1928.

Maternal grandmother said to have suffered from pulmonary tuberculosis, but pt. has never seen her; apart from this, no tbc. in family.

Pt. has always been perfectly well, except in fall 1927, when she had pertussis, lasting only 3 weeks, but of violent character. She was in bed a few days only, and the lesion subsided rather suddenly. Since then, pt. has been, perhaps, somewhat more short of breath than previously, when running about at play; otherwise, she has been perfectly well.

3 weeks ago, physician removed a foreign body (a toy-cartridge) from her left nostril. No after-effects from this.

She has been healthy and blooming till this morning, when she began to cough a little. During the afternoon, she was getting poorly, complaining of feeling ill, with increase of cough and onset of dyspnea. She was alone with a bigger sister, who put her to bed. At 8—9 in the evening, mother returned and noticed in addition to the dyspnea, a swelling of the pt.'s neck and head. As she was

afraid that the child had croup, she at once sent for her physician.

The family physician, Dr. *Max Søgaard Andersen, Troense,* has been kind enough to communicate the following date on the condition of the pt. at his arrival:

"I find the child markedly dyspneic, and bloated in the face, with moderate cyanosis. She is distinctly anxious and not quite clear. Tp. 37,2. P. 130, regular, soft. At the first glance, she reminded most of a case of very severe diphtheria. There is typical subcutaneous emphysema of the entire ant. surf. of the neck, extending a little way up into the cheeks, and around the right clavicle. Lung borders are not extended, being alike on both sides. No displacement of the heart. Perc. note over the sternum seems normal (i. e., without tympanitic sound). Slight tympanitic perc. note over entire right lung, with faint bronchial resp., and coarser and finer râles in upper portion of right lung. Left lung normal. Fauces normal. No other abnormalities on exam.".

"I made a puncture with a coarse cannula, but no air escaped from the pleural cavity — and I was greatly disappointed, I must admit. As I still felt sure of dealing with a case of lung rupture, and as I considered a permanent drainage to be the only chance for the child, I gave it an injection of coramin for stimulation, and drove it to the hospital immediately after".

At arrival to hosp., the physical exam. showed: The child is about moribund, dead pale, cyanotic, with faint, rapid pulse and labouring resp. (inspiration and expiration apparently equally impeded).

Rx. Inj. olei camphor. 2 cc. subcutan.

Ausc.: Tympanitic perc. on both upper post. surf., most marked on right side, where the subcutaneous emphysema started.

First, administration of *Oxygen inhalation* 5—10 minutes.

by which pt. straightened considerably,
and immediately after, performance of

Pleural puncture
on right side (with trocar).

Large amount of air evacuating at once. The trocar is connected with a rubber tube, the free end of which is placed under the water surface in a flask; and now the air is seen to bubble out freely, especially on expiration. For proof of the connection between the lung and the pleural cavity, under oxygen inhalation a glowing match is held above the water where the air is bubbling up from the tube — and the match flares up immediately. When this experiment is repeated after discontinuation of oxygen inhalation, the glow is at once extinguished.

The puncture has a most conspicuous effect on the pt. The cyanosis disappears, and the cheeks get a rosy hue; resp. is more quiet, pulse better, and pt. begins to say a few words.

After the trocar has been in for 40—45 minutes, a little blood begins to ooze out through the trocar; this is therefore removed.

After about 1 hour, the condition of the pt. begins again to be aggravated. Resp. becomes labouring, with heaving inspiration and difficult expiration, reminding of croup. Pulse becomes little and soft; and pt. is tossing about in her bed.

Pleural puncture repeated at 0,30 A. M.
„ „ „ „ 2,00 A. M.
„ „ „ „ 3,30 A. M.

Each puncture gives temporary relief. The child is coughing a great deal. With each puncture, there is a decrease in the amount of evacuated air, and at last the air is bubbling out only for 10 minutes.

On account of her cough

Rx. Syr. codeic fortior.
Half a teaspoonfull.

Tp. is now 38,5; P. 130, quite good.

The subcutaneous emphysema extends rapidly in all directions from the puncture site on the back.

Pt. sleeps a couple of hours, but gets worse towards morning, wherefore

Pleural puncture repeated at 5,15 A. M.

During the night

Rx. *Inj. olei camphor.*
1 cc. × 2.

After last puncture, pt. is more quiet, with deeper resp., and falls asleep. Tp. 37,8; P. 128, a little varying. No cyanosis.

April 25. Morning tp. 37,2. Urine: + sugar (strong).

X-ray exam.: A little air in right pleura. A few small spots and streaks around hilus. Extensive subcutaneous emphysema over entire chest and neck, extending down on the abdomen. Rather large liver; stomach completely distended with air.

April 26. 38,2/37,3. Urine: + sugar (trace).

X-ray exam. (cf. Fig. 29): Right-sided pneumothorax. Lateral of lung, from base to C.II, a 2½ cm. wide zone of air. Some infiltration of central portion of lungs. No particular change in the subcutaneous emphysema. Stomach is no longer filled with air.

April 27. 37,5/37,1. Urine ÷ sugar.

Pt. has been feeling quite well since the first night. Subcutaneous emphysema keeps rather unchanged.

Last night, pt. began again to cough, and was at once getting worse with difficulty of resp.

Rx. Syr. codeic. fortior.
½ teaspoonfull × 2.

Apart from this, she is bright and at ease, sitting up and playing.

April	28	29	30
Morning tp.	37,1	37,3	37,3
Evening tp.	37,9	37,9	37,4.

From then on, tp. normal.

April 30. Feels well, coughs a little. Appetite rather poor.

May 2. Coughs less. Feels well.

May 3. Cough almost completely disappeared, resp. normal. Subcutaneous emphysema subsiding gradually.

May 16. Feels well. No cough. No sign of subcutaneous emphysema.

May 21. Feels well. Up.

May 26. *Ausc.:* Normal findings. Discharged.

After-exam., Nov. 23, 1928: The child has been perfectly well since discharge; gaining in weight; no cough; no fever. Physical exam. shows a healthy-looking, well-nourished, vigorous child. *Ausc.:* No abnormalities. *X-ray exam.:* As on last exam., a few very small spots in region of right hilus; no other abnormalities (Plate not quite sharp, as it was difficult to get the child to hold her breath evenly.).

Sept. 9, 1929. Dr. *M. Søgaard Andersen* states that the child is perfectly well in every respect; especially, she has had no sign whatever of any lung lesion. *Pirquet:* ÷.

Sept. 2, 1931. Dr. M. Søgaard Andersen states that the child still is perfectly well; it has not shown any sign whatever of any lung affection.

Summary: Under coughing, acute onset of suffocating mediastinal and subcutaneous emphysema in a 5-years-old child, complicated with right-sided pneumothorax. Pt. moribund on adm. to hosp. The life of the child is saved through repeated punctures, oxygen inhalation and stimulation. After-exam.: Perfectly well during past 3¼ years. No sign of tuberculosis.

CASE 49.

Female, 23 years, servant.

Svendborg Hosp., Febr. 2, 1929—March 22, 1929.

No tuberculosis in family. Past history of good health.

Nearly 1 year ago, at Whitsuntide, May 1928, pt. was cycling to church. She had to cycle fast in order to come

in time, and she was rather short of breath when she got to church. While sitting in the church, she had *suddenly* a violent pain in left side of chest. The pain was most severe the first half hour, and was then subsiding. The next 10 days, she was readily short of breath when she worked, and felt a stitch in the left side of chest. Since that time, she has been perfectly well and capable of work, without any local or general symptoms whatever.

2 weeks ago, she went to bed on account of a cold. She was very ill a couple of days with coryza, headache and cough; tp. was slightly elevated. She was examined by her physician, Dr. *Erik Aagaard,* who referred her to Dr. *A. Paaby* for *X-ray exam.,* which showed total left-sided pneumothorax.

On adm., Pt. is rather thin and delicate, but looks healthy otherwise. Tp. 37,2. *Ausc.:* Heart findings normal; heart partly lung-covered. Right lung normal. Resonant perc. and faint bronchial-amphoric resp. over entire left lung, most marked over lower part. No dullness or râles. *X-ray exam.* (Jan. 28) *(cf. Fig. 12):* Left-sided pneumothorax. Lung is partly compressed. From apex an adherence upward to C.I. Right lung shows rather accentuated markings (probably congestion) and enlargement of hilus.

Tp. perfectly normal throughout stay in hosp.

Febr. 9. *X-ray exam.:* Quite the same findings as on Jan. 28. No change in pneumothorax.

Febr. 10. *Ausc.:* Circumference of left half of chest 1 cm. larger than right side, and lagging a little on resp.; post. border at C.XII. Resp. faint, some places with amphoric note. Right lung normal. *Pirquet:* ÷.

March 5. *X-ray exam.:* Some expansion of left lung.

March 14. Up.

March 22. *Ausc.:* Same findings as on adm. *X-ray exam.:* Lung expanded a little more than on March 5.

Oct. 1, 1929. According to communication from Dr. *Erik Aaagaard, Gislev,* pt. is feeling perfectly well in every

respect, and doing hard farmwork. *X-ray exam.* (Sept. 26, 1929): No abnormality. No sign of pneumothorax.

Sept. 2, 1931. Dr. *Erik Aaagaard* states that he has again examined the patient. She is well and healthy. 2 months ago she gave birth to a child, at full term, after normal and uneventful pregnancy; and now she is working hard. Physical exam. reveals no evidence of any illness whatever, especially no sign of tuberculosis or other lung lesion.

Summary: Acute onset of left-sided pneumothorax with slight displacement. Pneumothorax not discovered till 8 months after onset. Slow expansion of the lung. After-exam. 3¼ years after onset of illness: Perfectly well. No sign of tuberculosis.

CASE 50.

Female, 26 years, Master of Arts.

Kommunehosp., Dep. II, June 6, 1929—June 7, 1929.

No tuberculosis in family. Past history of good health.

Pt. has been abroad the last couple of months, and has been perfectly well till 4 weeks ago. At that time, she was staying in the mountains and was climbing a great deal, which she stood very well. The last day in the mountains, she was on a climb — ca. 1900 m. above the sea, — without feeling anything unsual. Next morning, that is,

4 weeks ago, she was getting very short of breath as soon as she got up, and had palpitation and cough on slightest exertion. She had no real pain, but some kind of soreness of the breast when moving about or coughing. No expect. No rise of tp. The first week, pt. became dyspneic on slightest exercise, even speaking. Dyspnea subsided by and by; and during past 3 weeks her symptoms have kept fairly stationary. Now she is really troubled only with dyspnea on more vigorous exercise; and sometimes she gets some cough from exercise. Never any rise of tp. or pain.

On adm.: Looks healthy. Tp. 37,7; P. 90. *Ausc.:* Left side normal. Right side: Slightly tympanitic perc. over

entire right lung. No dullness. Resp. markedly impaired over right apex and post. surf. to spine of scapula, inaudible over rest of lung. No particular change in vocal phenomena. No râles. Ictus in IC.V, 2 cm. outside midclavicular line; dullness from this point to 2 cm. left of sternum. $P_{II} = A_{II}$. Heart-sounds clear, action regular. *X-ray exam.:* Right-sided total pneumothorax, with complete compression of lung. No abnormality noticeable in left lung, but pt. has not held her breath during exposure. Some displacement of heart to left.

Aspiration with Dieulafoy's syringe — evacuation of 1300 cc. of air.

Pt. became somewhat dyspneic and cyanotic, but straightened up soon. Resp. more audible after aspiration.

June 7. Morn. tp. 36,8; P. 84. *Ausc.:* Resp. markedly suppressed on right side, but still audible clear down to base.

June 8. *X-ray exam.:* Partial expansion of right lung, but still a 1 cm. wide rim of air along chest wall, a little wider in upper part of field. No abnormality in lung itself. Leftward displacement of heart disappeared.

As pt. was merely stopping here on her way through, she was discharged on June 7.

Letter from pt., Jan. 6, 1930: *X-ray exam.:* (June 18, 1929) showed a coat-formed layer of air around the lung. Pt. has been perfectly well during past ½ year, except for a little fatigue during the first 1½ month. Has been examined several times during this period, and was always found healthy.

Letter from pt., Sept. 13, 1931: Perfectly well in every respect. Examined by her physician, who could not find any abnormality whatever in her lungs.

Summary: Acute onset of right-sided pneumothorax with moderate displacement. Adm. 4 weeks after onset. Aspiration with good effect. Perfectly well during past 2¼ years. No sign of tuberculosis.

CASE 51.

Male, 38 years, tobacco worker.

Kommunehosp., Dep. II, July 10, 1929—Aug. 24, 1929.

Parents and 10 brothers and sisters living and well. 1 cousin died of pulmonary tuberculosis. Past history of good health.

Yesterday morning, without preceding exertion, *sudden stitch* in left side of chest around left clavicle. As stitch was increasing during the following hour, he was taken to hosp., where no abnormality could be demonstrated; so he was taken back home. During the day, some aggravation of the stitch and some sensation of tension in left half of chest. No cough or palpitation.

On adm., at 1,30 A. M.: Pt. is not particularly distressed. Tp. 37; Resp. 19; P. 74, regular. No particular dyspnea; resp. about normal, but some shortening of inspiration on account of stitch. Pt. is rather pale and thin, perhaps slightly cyanotic. *Ausc.:* Pt. lying on right side. Marked distension of entire left half of chest, which does not take part in resp. Tympanitic perc. over entire left ant. surf. and over sternum to right sternal border. Lung borders C.VII and post. C.XI—XII. Resp. inaudible. Heart displaced to right; ictus at upper point of epigastrium; right border obscure. Right lung normal.

July 10. 37,5/37,2. P. 76. Complains a good deal of stitch in left axilla.

July 11. *X-ray exam.:* Left lung completely compressed and left pleural cavity filled with air. Moderate displacement of heart to right. No effusion.

Less pain the last 24 hours. Resp. 24. *Ausc.:* Left half of chest is still considerably distended and lagging. Resonant perc. note over entire left lung. Resp. is inaudible, but a few metallic sounds are heard. Heart sounds most distinct at ensiform process. *Pirquet:* +.

July 16. Feels well. No pains.

Tp. perfectly normal throughout stay in hosp.

July 25. *X-ray exam.:* Left lung is less compressed, and heart is less displaced to right.

Aug. 1. Resp. perhaps a little more audible at base.

Aug. 3. Up.

Aug. 8. *X-ray exam.:* Lower part of left lung more expanded, without reaching lateral wall of chest; no change in upper part.

Aug. 14. *Explor. Puncture,* on left side in IC.VIII in angular line. Pressure was ÷ 8 and ÷ 4 cm. H_2O. Aspiration of ca. 1000 cc. of air, whereafter a little cough. Pressure is then ca. ÷ 20 and ÷ 18 cm. H_2O.

Aug. 16. *X-ray exam.:* Left lung has expanded considerably.

Aug. 20. Perc. is now the same on both sides. Resp. audible over entire left lung, but faint in supraspinata and at base post.

Aug. 23. *X-ray exam.:* Left lung seems completely expanded throughout, perhaps lacking a little bit at upper part of apex; still, distinct progress since last exam.

Aug. 24. Discharged.

After-exam., April 9, 1930: Perfectly well since discharge, with exception of occasional slight stitch in lower part of left axilla on deep resp. *Ausc.:* Occasional friction sound in this region. Otherwise normal findings.

After-exam., Nov. 25, 1930: Perfectly well, with exception of occasional slight discomfort over precordium. *Ausc.:* Lung borders distended on back (C.XI), otherwise no abnormalities. *X-ray exam.:* Both hilus shadows are rather dense, mostly of fibrous appearance with rather dense spots (up to pea-sized), more suggestive of calcifications than of vessels. *Right* hilus shadow is undoubtedly enlarged upwards, and here is an almost walnut-sized, rather conspicuous, cap-shaped density, rounded in outline and regular, which is very suspicious of a lymphoma. Slight veiling of right apex with small linear density and a rather unquestionable increase of perihilar lung markings, partly

as shadows of vessels but also, as far as can be made out, arranged as bronchi; perhaps this may be due to beginning calcification of bronchial cartilage, which is strikingly conspicuous in several of the smaller bronchi. There is a very slight blurring of sinus phrenicocostalis but otherwise no pleural adhesions at the base. *Left* apex is diffusely veiled, much more than on right side. No sign of definable glands in the hilus shadow, which is not enlarged. There is possibly a hardly pea-sized calcification in lower part of hilus shadow. Lung markings similar to those on right side, but vessels rather less conspicuous. On the whole, the lung markings are rather somewhat clear on both sides; intercostal spaces rather wide. Diaphragmatic arches rather low (C.XI). Aorta shadow somewhat stretched, and arcus slightly prominent — all suggestive of slight emphysematous changes. (Dr. *Jens Nielsen*).

After-exam., Sept. 11, 1931: Perfectly well since last. *Ausc.:* Slight emphysema; otherwise no abnormalities. *X-ray exam.:* No abnormalities, excepting a roundish, well-defined, density (size of large hazel-nut) in right hilus, probably enlarged lymph gland. (Dr. *E. Collin*).

Summary: Acute onset of left-sided pneumothorax with moderate displacement. Adm. on day of onset. 35 days later, aspiration with good effect. After-exam.: Perfectly well during past 2 years. No sign of tuberculosis.

OCT 8 1953

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